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Special Article

FEEDING PROBLEMS OF YOUNG CHILDREN OF PRE-SCHOOL AGE

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The prime biological necessity for eating naturally implies that any disturbance of this function in a child will be looked upon with anxiety by the parents. Hence, children of pre-school age are often brought to the doctor with the complaint that 'my child won't eat'. This does not necessarily mean that the child is suffering from anorexia, i.e. lack of appetite, as the term is generally used in adult practice. It is the general experience that 'my child won't eat' refers to a number of feeding disorders, varying widely in their causation, mode of onset, and pattern of behaviour. Thus, on being asked to qualify the main complaint, parents may state that the child 'won't eat what is good for him', or that he is finicky or fussy or capricious about his food, or that he will only eat if his mother tells him stories. Other departures from normal feeding behaviour, as observed by parents, will be indicated by the following remarks:

'He won't eat a thing.'

'He won't touch his food.' 'He hardly touches his food.'

'He's maddeningly slow about his eating.'

'I have to coax him to eat.' 'I have to force him to eat.'

'He won't eat his vegetables.' 'He only wants to eat sweets.'

'I'm afraid he will get ill.' 'I'm afraid he's sickening for something.'

These 'cries from the heart' will be familiar to all practitioners. Listened to carefully, without interruption, they provide, like all medical histories, valuable diagnostic clues. They also afford a much needed opportunity for a harassed mother to unburden herself of her pent-up feelings.

Common Features

Though varying in aetiology, feeding disorders tend to have certain features in common. In the first place evidence of ill-health (except in the small group to be described later), is rarely found on physical examination. The nutritional state, too, will be found to be far better than one would have anticipated from the harrowing tales of 'not eating' described by the parents. The third and most important common feature is that the *anxiety* exhibited by the parents and family circle appears to be out of proportion to the child's apparent well-being and good health. It is often a degree of anxiety one would normally expect when a child is seriously ill. The anxiety may be revealed by the intense preoccupation with the child's feeding habits. Other symptoms and other topics may be referred to, but sooner or later the discussion will revert to the central theme.

The Significance of Anxiety

It is important to investigate the reasons for excessive parental anxiety so that parents can be reassured and their fears resolved, by a clear insight into their problems.

The anxiety is an expression of one or several *fears* which may be overt, i.e. directly expressed, or discovered only by more detailed questioning. In the preface to *Of Human Servitude*, Spinoza says: 'For where a man is prey to his emotions, he is not his own master, but lies at the mercy of fortune, so much so that he is often compelled, while seeing that which is better for him to follow that which is worse'.

There is the obvious fear that the disturbance is a manifestation of ill health, that it will leave the child deformed or retarded, that it will inhibit his growth, that he will become a chronic invalid, or worst of all, that he will die of it. There is the deep anxiety and hurt felt by a mother that by refusing to eat the food she has prepared for him, the child is rejecting the love she bears for him.

Anxiety is particularly common in the mothers who have a feeling of guilt about their children. The child might not have been wanted, or its arrival might have interfered with the mother's professional or other interests. The mother compensates for this feeling of guilt by excessive indulgence. Any illness or upset shown by the child may be interpreted as a form of punishment meted out to the mother for past misdemeanours, real or imaginary.

Parental anxiety is likely to be marked where the child is particularly precious. There is the only child, the only boy in a family of girls, the first grandchild in a large family, the child born after many years of marriage; there is the child born to middle-aged parents, long after a previous birth, and the first child born after one or several tragedies. These children are candidates for behaviour problems of all kinds, and particularly of feeding problems.

The child's reaction to abnormal parental anxiety is familiar to all conversant with common behaviour problems. Should he at any time imagine or suspect that he is not receiving his quota of parental love and attention, there is a sure and certain way of improving his situation — he has merely to become a 'feeding problem'. He may do this by creating a disturbance at table by refusing food altogether, by merely nibbling at a previously favoured food, or by eating only under conditions which will disrupt the household routine. He may use his 'not eating' as a direct form of blackmail, promising to eat only after certain demands have been exacted. If there is parental or family disharmony, so much the better; if one will scold, another will rush to his defence, but he knows that when the tumult and the shouting dies, he will still emerge as the central figure on the stage.

SOME COMMON FEEDING PROBLEMS

It is not easy to classify the feeding problems of young children. A broad classification would include: those associated with acute and chronic ill-health, dietary faults due to ignorance about the fundamental nutritional requirements of children and about the preparation of food, and psychological disturbances. It should be remembered, too, that several feeding problems can, and do, frequently exist together in the same child.

1. *Feeding Disorders Associated with Acute or Chronic Ill-health*

Only a small proportion of children who 'won't eat' belong to this group and the cause is usually self-evident. The common example is of the child who goes off his food at the onset of an acute infection. Here true anorexia is present, but it is rarely the most prominent or only symptom, being seen in its true perspective as the diagnosis becomes established. In chronic ill-health, it may take a little longer to realize that the anorexia is merely one symptom of a particular disorder. When pica or perverted appetite is a feature of the feeding problem in this age group, one ought to look for evidence of iron-deficiency anaemia. Lanzkowsky[1] has shown that correction of the anaemia in these children produced a rapid and dramatic cure of the pica, usually within 1 - 2 weeks of the beginning of the therapy.

2. *Faulty Notions about the Feeding or Nutritional Requirements of Children*

In my own experience many of the children who 'won't eat' belong to this category. Becoming a parent does not necessarily bring with it a comprehensive knowledge of child-care or of nutrition. In addition, there are the obvious limitations imposed by socio-economic factors such as poverty, overcrowding, the lack of proper facilities for the cooking and preparation of food, and the necessity for the mother to go out to work.

Within those limitations, one of the commonest problems in this group concerns the child who 'won't eat what's good for him'. This, of course, means what mother father, aunt, or person-in-charge considers is good for him. With the best intentions in the world, what is thought to be good for him may not be consistent with accepted nutritional standards, or with the child's own concept of what is good for him. (The philosophy of authority is closely bound up with the "what's good for him" outlook, but only the nutritional aspect of the subject will be referred to here.)

What a mother considers good for her child will, of course, vary enormously. Generally speaking, where finances allow, the average mother will choose a reasonable diet for her children. Difficulty arises when the child has marked likes and dislikes which do not run parallel to parental concepts. The mother may be convinced about the efficacy of a food or foods for which the child has no liking. Or, conversely (and perversely), the child may show a preference for foods which the mother does not consider good for him. Differences may also arise about the quantity and quality of the food, i.e. the amount of food eaten, and the way it is prepared and served.

The best way to assess the situation in the 'won't eat what's good for him' group (apart from living in his home for a while), is to investigate *in detail* the child's daily feeding routine and to make a careful study of his eating habits, particularly of his likes and dislikes. Many studies have shown that this type of child, if given a free choice, would select a good well-balanced diet. On the other hand, if the child's choice of food conflicts with that of the parents, disaster threatens.

It is a tragi-comic situation that is encountered in conversation with these parents: 'Does he like meat?' 'He can eat it twice a day'. 'Does he like fish?' 'Yes'. 'Eggs?' 'Yes'. 'Fruit?' 'Yes'. Then there is a pause, and there follows the inevitable, 'But doctor, he won't eat his vegetables', or 'he won't drink up all his milk' as the tragic denouement.

The resultant psychological explosion takes two forms: (*a*) Parental anxiety because the child bilks at food considered essential for his health, and (*b*) disturbance of the child's natural good appetite by the tense situation produced by quarrelling, bullying, blackmail, and resentment. 'You can only have a second helping or enjoy your favourite pudding if you . . . ', sums up the situation. If the child rebels, as so often happens, mealtimes become battles involving not only mother and child, but often other members of the family as well. The inevitable result is that mealtimes become associated with unpleasantness and a general state of tension.

A good illustration of the 'won't eat what's good for you' mentality applies to the common dietary fault of attaching too much value to *boiled vegetables* as an essential item in the diet of young children. Now, while most children like well-prepared salads of appetizing appearance, few are fond of boiled vegetables, particularly when, as so often happens, they are badly prepared, i.e. mixed indiscriminately together to give the child the 'maximum amount of goodness and vitamins'. But the poignant cry of 'he won't eat his vegetables', tends to make this not-so-essential item the most precious commodity in human diet.

Milk is also a food about which misunderstanding often prevails. (This statement applies to economically well-off children and not to those who are kwashiorkor prone.) Assuming that a well-balanced diet is accepted, 1 pint of milk a day is ample for children of the pre-school age, but many mothers expect their children to drink twice that amount. If a child is not fond of milk, or if he objects to boiled milk, which his mother insists on, a feeding problem is invited.

OTHER DIETARY FAULTS

Other food fads and fancies may be revealed by careful interrogation. Apart from the well-known 'diets' which enjoy the fashion of the moment, there are many mistaken notions about food which will be familiar to practitioners of experience. As examples of common fallacies in this part of the country, there is a popular notion that eggs are harmful to small boys if given more than once or twice a week; the harm is not related to cholesterol but to their supposed aphrodisiac effect. In the Cape Flats, folklore maintains that meat 'brings worms', and numbers of young children may be deprived of meat for this reason.

Henson,[2] in a fascinating article on false medical beliefs.

discusses some of the false beliefs concerning food encountered in the course of his work at the Health Centre, Grassy Park. Among these are: that too much meat and/or bread causes worms, that squashes contain valuable foodstuffs, and that lemons dry up the blood. Guavas are said to be unhealthy because they cause appendicitis, and in winter time tomatoes are dangerous to give to children. Popular belief also avers that *maasbankers* (a kind of fish) are not fit for decent people to eat, though Henson points out that these fish are cheap, rich in vitamin A, and delicately flavoured when cooked fresh. The various 'diets' which are so popular have already been referred to. Therapeutic dieting by adult members of the family, as recommended for obesity, peptic ulcer, hypertension, and diabetes, may also be disturbing to the child by setting a bad example in eating habits. Social customs and national dietetic habits can create difficulties, e.g. when parents maintain a conservative attachment to traditional dishes, which are not always acceptable to the young.

A common dietary fault concerns the *preparation* of food. Good food, well prepared and pleasantly served, is not only the privilege of grown-ups, but parents regularly fail to understand this. A small child is often expected to eat a 'mush' which would nauseate the delicate palates of his elders.

PSYCHOLOGICAL ASPECTS OF THE 'CHILD WHO WON'T EAT'-PROBLEM

Some of the psychological factors of this problem have already been mentioned. They include the element of parental anxiety and its effects, conflict between parent and child in the feeding situation, and conflict between parents — expressed either in general marital disagreement, or in respect of the child's feeding habits. The issue becomes further complicated when, as often happens, other members of the family become involved. *In general, any situation which causes tension in the home may be the cause of poor or disturbed eating habits.* Common speech is full of allusions to the action of the emotions upon the appetite or digestion — 'Too upset to eat', 'too frightened to eat', 'too tense to eat', 'too excited to eat'. On the other hand everyone agrees that relaxation and absence of tension, as exemplified by an atmosphere of love and good fellowship, is conducive to healthy appetite and the pleasurable enjoyment of food. 'Now good digestion wait on appetite and health on both' says Shakespeare. 'Better a handful of herbs where love is', says *Proverbs*, 'than a stalled ox and hatred therewith'.

Among the causes of tension not yet referred to, is the situation produced by *sibling rivalry*. This is usually precipitated by the arrival of a new baby. The child who has reigned supreme, perhaps for several years, suddenly has to share with a hated rival. Refusing to eat, eating only if bribed or coaxed, eating only if fed by mother — all these are attention-drawing devices which rarely fail.

Rigid discipline is another important cause of tension. The withdrawal of a favourite food unless or until the less favoured one is accepted, has been mentioned. The *slow eater* too, is often labelled a 'won't eater' by a demanding parent, though basically there is nothing wrong with his appetite. Another form of feeding discipline concerns a *too rigid schedule*. The child, with hardly any warning and at a given signal, is expected to drop his toys or stop his play, run and wash his hands, and present himself at table. There he must act the Little Lord Fauntleroy, complete with correct table-manners, manipulating knife and fork with dexterity, and consuming all the food set before him — pleasantly and efficiently. This, of course, is an exaggerated picture, but modifications are common, and tension is the result.

One way of demonstrating the effect of tension, is to enquire into the child's feeding behaviour when there is a *minimum* of tension present. This situation fortunately, does occur at times, such as when the family is on holiday, or enjoying a picnic together at the sea-side or in the country. There is plenty of fun and adventure with the thrill of cooking and preparing a meal in the open at a picnic. Discipline is relaxed, table manners are not *de rigueur* and there is no argument about what should or should not be eaten. At such times little Tommy, the 'won't eater', cannot get enough, often making a pig of himself. At a birthday party or a visit to friends, the impossible Tommy will behave with the utmost decorum, and his appetite and behaviour at table would surprise and gladden the hearts of anxious mothers, grannies, and aunts — if they could see without being seen or heard!

In assessing the significance of the psychological element in these problems, the opinion of the family doctor, by virtue of his intimate knowledge of the child and the family background, is of particular value. His experience should be useful in preventing a too-hasty label of 'behaviour problem' before the other causes previously mentioned have been studied and investigated.

CONCLUSION

Treatment of the 'child who won't eat' involves far more than mere physical appraisal of the child. It necessitates a knowledge of the food requirements of children of various ages and an understanding of the normal and abnormal feeding behaviour of children at the various stages of development. In addition, the treatment of these children requires an appreciation of how emotional problems may affect the appetite and eating habits of young children of the pre-school age. Finally, it should be remembered that the child who is a problem-eater is likely to have problem parents who need to be treated with sympathy and given guidance and reassurance.

SUMMARY

In the clinical approach to the problem of feeding disorders in young children, the following regime is suggested:

1. Look for evidence of ill-health.

2. Investigate in detail the child's feeding schedule throughout the day, noting *when* he eats, *what* he eats, *how* he eats, and whether he feeds himself. Enquire into the child's likes and dislikes with regard to food, and whether these accord with parental notions. If possible, be present at a meal or two. This will give some idea of how the meals are prepared and served, and will give an indication of the family relationships and discipline at table. Find out, or observe if possible, whether the child is a 'slow eater'.

3. Look for emotional disturbances causing tension, e.g. sibling rivalry, parental disharmony, excessive disci-

pline. Learn to recognize and appraise the anxiety and tension associated with the 'precious child'.

4. The true nature of the problem, after it has been unfolded, should be explained to the parents. Physical disturbances should be treated. If the feeding programme has been wrong, it should be pointed out to the parents and corrected. Other faulty approaches in child management, if present, should also be attended to. Lastly, seek the cause or causes of the parent's anxiety and allay these by frank discussion and reassurance.

REFERENCES

1. Lanzkowsky, P. (1959): Arch. Dis. Childh., **34**, 140.
2. Henson, J. (1950): S. Afr. Med. J., **24**, 203.

THE EFFECTS OF ADRENALECTOMY ON NON-DIETARY CIRRHOSIS IN RATS*

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In discussing this subject certain views based on earlier experiments[1] were modified before the effects of adrenalectomy on egg-yolk cirrhosis were dealt with.

Previously it was thought that a renal lesion was essential to the development of egg-yolk cirrhosis and that histamine liberation might be directly involved in its pathogenesis. Neither view had been substantiated. Repeated intravenous administration of egg yolk alone to 17 rats for periods of 18 - 130 days caused cirrhosis in 15, and a potent histamine liberator, 48/80,† failed to cause hepatic fibrosis in any of 8 animals given this substance intraperitoneally for periods of 16 - 57 days.

Contrary to original expectation, egg-yolk cirrhosis had also been proved a self-limiting lesion, and animals apparently developed a tolerance to egg yolk. In 15 animals, observed for periods of 50 - 145 days, regularly repeated injections failed to produce any increase of the fibrosis or signs of portal hypertension. In addition, it had been possible to prevent or reduce the cirrhosis by giving either cortisone (5 - 10 mg.) or 'phenergan' (5 mg.) whenever egg-yolk injections were made. These findings related, however, to only 3 animals.

This non-dietary cirrhosis was furthermore not specific to egg-yolk, and an essentially similar lesion had followed repeated intravenous injections of chick-embryo extract (7 of 8 animals), egg white (3 animals) and horse serum (1 of 2 animals). The common factor in these experiments is the injection of large amounts of foreign proteins, and though this is not related to current ideas on the pathogenesis of human cirrhosis, it has experimental precedent in the work of Wells[3] and of Longcope.[2]

The modifications in this cirrhosis following bilateral adrenalectomy were then described. The adrenals were removed in two stages, and the animals maintained on 1% salt water after the second adrenalectomy. The second gland was removed usually 1 - 3 weeks after the first in 11 controls, to whom no egg yolk was given, and after an interval of 23 - 33 days in 12 experimental animals. Liver biopsies were taken at the time of the second adrenalectomy in control and experimental groups, and repeated at intervals of 7, 14, 21, 35 and 68 days in some of the controls. In all control animals, bilateral adrenalectomy caused some weight loss and hepatic atrophy was present in 4 that died, but in no control was evidence of hepatic fibrosis found in postmortem or biopsy material.

Unilateral adrenalectomy in the group of 12 experimental animals did not influence the development or progression of the cirrhosis: however, in every case bilateral adrenalectomy produced significant effects which appeared to show some correlation with the severity of the existing hepatic changes. In the early stages when changes were mild and confined to eosinophile infiltration and regenerative activity, as in 4 of the experimental group, the animals survived 25 - 122 days, gained weight, and developed Laennec-type cirrhosis in the usual way. This cirrhosis was not significantly more marked than in rats with intact adrenals, but in 1 of the 4, ascites appeared and re-accumulated despite repeated withdrawals of fluid. It was admitted that incomplete adrenalectomy could be responsible for the long survival of this group of 4 animals, and evidence of this was subsequently obtained microscopically in the long-term survivor which developed ascites.

The remaining 8 animals survived the second adrenalectomy and the continued egg-yolk injections for only 7 - 9 days. Death was preceded by precipitous weight loss and refusal of food and water. In these 8 rats, liver biopsy at the time of second adrenalectomy showed isolated liver-cell necrosis and minimal fibrosis, but at postmortem a remarkable transformation in the microscopical appearances in this short survival period of a few days after bilateral adrenalectomy was found. Fibrous bands were increased in width and number, liver-cell necrosis was widespread (but unaccompanied by fatty change and never of focal coagulative or so-called massive type), liver-cell nuclei were large and hyperchromatic, and this feature, together with increased mitotic activity, gave to the hepatic tissues an appearance of anaplasia. Eosinophile infiltration in the portal tracts was intense, sinusoid congestion pronounced, and Kupffer cells apparently engaged in tremendous phagocytic activity in a manner not previously seen in experiments with egg yolk.

The differences before and after bilateral adrenalectomy are as great as can be imagined, and yet, with the exception of two features, the intense Kupffer-cell activity and the more widespread liver-cell necrosis, all the changes are simply an intense exaggeration of those seen in animals with intact adrenals. Clearly, further experiments are needed to show whether bilateral adrenalectomy alone is responsible for these changes or whether, as in these experiments, egg-yolk injections must be continued for the changes to occur.

REFERENCES

1. Campbell, J. A. H. (1960): S. Afr. Med. J., **34**, 336.
2. Moon, V. H. (1934): Arch. Path. (Chicago), **18**, 381 (quoting Longcope).
3. Wells, H. G. (1920): *Chemical Pathology*, p. 576. Philadelphia: Saunders.

* Abstract of a paper presented at Research Forum, University of Cape Town, 16 November 1960.

† Kindly supplied by Dr. A. C. White of Burroughs Wellcome & Co., London.

Belangrik. Die aandag van lede word gevestig op 'n verandering van die inligting gepubliseer in die *Tydskrif* van 11 Februarie (**35**, 116). Die vergaderplek van die Buitengewone Algemene Vergadering van die Vereniging is verander na die **Festival Hall, Maitland-hotel, Maitlandstraat, Bloemfontein,** en sal *nie* in Johannesburg gehou word nie. Hierdie vergadering is saamgeroep om die instelling van die Mediese Diensplan in al die Provinsies van Suid-Afrika te oorweeg, en sal om 10 vm. op 8 Maart 1961 gehou word.

Important. Will members please note that contrary to the information published in the *Journal* of 11 February (**35**, 116), the venue for the Extraordinary General Meeting of the Association will be the **Festival Hall, Maitland Hotel, Maitland Street, Bloemfontein,** and *not* Johannesburg. This meeting has been called to consider the implementation of the Medical Services Plan in all Provinces of South Africa, and will be held on 8 March 1961 at 10 a.m.

VAN DIE REDAKSIE : EDITORIAL

SPONTANE MISKRAAM

Die veelvuldigheid van vraagstukke wat in verband staan met die probleem van spontane miskraam, is betreklik onlangs weer deeglik bespreek deur 'n verteenwoordigende groep deskundiges.[2] Daar is toe aangetoon dat ten spyte van die feit dat die toestand so dikwels voorkom, daar baie fasette van die probleem is waaroor ons nog heeltemal onkundig is. 'n Poging is egter aangewend om die feite waarmee ons wel bekend is, in oorsig te neem.

Dit wil voorkom of daar ongeveer 'n 15%-kans is vir enige swangerskap om in 'n miskraam te eindig. Om te weet of dit die gevolg is van genetiese abnormaliteite wat patologiese ovums voortbring, en of dit te wyte is aan omgewingsfaktore wat òf patologiese ovums òf afwykende funksie van die uterus veroorsaak, is somtyds baie moeilik. Diegene wat meen dat genetiese abnormaliteite aan die wortel van die kwaad lê, wys daarop dat abnormale ovums in 52 - 73% van gevalle aangetref word. Alle gevalle van miskraam kan egter nie op hierdie grondslag verklaar word nie.

In die geval van 'n normale swangerskap is reis nie as sodanig 'n etiologiese faktor by miskraam nie. Ernstige trauma is egter wel 'n faktor, en toevallige snykundige behandeling gedurende swangerskap kan ook 'n rol speel. Om die rol van emosionele faktore onder gekontroleerde omstandighede vas te stel, is baie moeilik. Sommige deskundiges gee egter tog aan die hand dat psigiatriese hulp verleen moet word in gevalle waar miskraam herhaaldelik voorkom. Daar word gesê dat ongeveer een derde van alle miskrame (en twee derdes van alle voortydige bevallings) veroorsaak word deur funksionele afwykings van die miometrium, aangesien saamtrekbaarheid van die uterus afhang van en varieer volgens die konsentrasie van die hormone van die ovarium, en van estrogeen en progesteroon, of van epinefrien wat vrygestel word deur weefselbeskadiging of psigiese skok. Dit mag wel moontlik wees dat liggaamlike en emosionele trauma die endokrien-omgewing verander asook die stowwe van die miometrium wat by saamtrekbaarheid gemoeid is, sodat sametrekkings van die uterus, bloeding van die buitenste vrugvleis, en miskraam by vatbare persone ontstaan.

Die voorkoms van abnormaliteite by miskraam-fetusse is baie hoër as by babas wat op tyd gebore is. Dit mag dus verleidelik wees om hieruit af te lei dat abnormaliteite verantwoordelik is vir die vroeë beëindiging van die swangerskap. In gevalle van ernstige abnormaliteite mag dit wel die geval wees, maar dit is moeilik om te bedink hoe 'n plaaslike abnormaliteit, soos byvoorbeeld 'n gesplete verhemelte, 'n miskraam kan veroorsaak. Dit is gevind dat die voorkoms van misvormde kinders (in 'n groep kinders wat gebore is na 'n dreigende miskraam voorkom is) nie groter is as in 'n groep waar daar geen miskraam was nie. Dit dui daarop dat abnormaliteite van die fetus waarskynlik nie in verband staan met daardie dreigende miskrame wat tot tyd voortgaan nie.

Baie aandag is al geskenk aan die probleem van die moontlikheid van meer miskrame nadat 'n miskraam voorgekom het. Skattinge van die moontlikheid varieer van 6 tot 20% met 'n gemiddelde van 10%. Dit is moeilik om die voorkoms van herkenbare miskraam in enige samelewing te bereken, aangesien onherkende vroeë miskrame nie bygereken kan word nie. Dit word gewoonlik (alhoewel dit nie noodwendig altyd korrek is nie) aanvaar dat 'n vrou wat een of meer miskrame gehad het, meer blootgestel is aan nog 'n miskraam as 'n vrou wat dieselfde aantal voltydse swangerskappe gehad het. Die syfers wat gewoonlik in hierdie verband aangehaal word, is die van Eastman wat op Malpas se metode hereken is, dat, as die totale voorkomssyfer van miskraam 10% is, dan is die kanse om 'n miskraam na een vorige miskraam te hê, 13% ; na twee, 37% ; en na drie 84%. Hierdie berekeninge is waarskynlik te hoog. Hulle stem nie ooreen met die empiriese bevindinge, wat nou beskikbaar en baie laer is nie, en wat verbasend konstant bly as die verskillende monsters van die samelewing wat gebruik is, in gedagte gehou word. Op grond van hierdie berekeninge wil dit voorkom dat die kanse op 'n miskraam na 'n vorige miskraam ongeveer 20% is, met slegs 'n geringe vermeerdering tot 25% soos die aantal miskrame toeneem. Hierdie syfer, 25%, is belangrik (in teenstelling met 84%) by die bepaling van die waarde van voorbehoedende behandeling in sulke gevalle.

Die neiging vir miskrame om meer by sommige vrouens voor te kom as by ander, skyn aan te dui dat sekere van die oorsake by herhaling kan voorkom. Dit is egter glad nie seker dat die neiging 'n biologies-anderse groep verteenwoordig wat konstitusioneel nie in staat is om swangerskappe te behou nie. Die neiging kan ook die gevolg wees van blywende faktore in die moeder se omgewing, van permanente gevolge van 'n tydelike omgewingsfaktor, of van moederlike of fetale gene waarvan sommige dominant en andere resessief kan wees. Daar bestaan baie min bewyse oor watter, indien enige, van hierdie meganismes, werklik etiologies beduidend is.

1. Danforth, D. N. (1959): *Clinical Obstetrics and Gynaecology*. New York: Paul B. Hoeber Inc.

CHROMOSOMES STILL ON THE MARCH — AUTOSOMAL TRISOMY

Deviations from the normal diploid chromosome number of 46 in man (aneuploids) have been recently described in a rather surprisingly large number of conditions associated with particular congenital defects. Abnormalities affecting the sex chromosomes include monosomy (as in gonadal-dysgenesis-with-female-body-form) and trisomy (as in Klinefelter's syndrome). Aneuploidy affecting the autosomes (non-sex chromosomes) has so far been in the nature of trisomy only.

Autosomal polysomy has seldom been observed in animals ; most instances have been recorded in *Drosophilia* spp.[1] In *D. melanogaster*, monosomy and trisomy for

chromosome no. 4 produces virtually no deviation from normality, though flies tetrasomic for this chromosome are inviable. In view of the apparent rarity of animal polysomy, the recent discovery of five different varieties of human autosomal trisomy seems surprising.* The probable reason for this discrepancy is that in man studies have been confined to phenotypically abnormal individuals selected from large populations. No comparable study has been performed in any other species.

The first recorded human autosomal trisomy was found in mongolism in 1959 by three separate groups of workers,[2-4] in which condition chromosome no. 21 was affected. Patau and co-workers[5] described trisomy for chromosome no. 15 in a mentally defective girl, who had trigger thumbs, polydactyly, capillary haemangiomata, harelip, cleft-palate, apparent anophthalmia, and a heart defect. Edwards and associates[6] described trisomy in chromosome no. 17 in association with odd face, webbed neck, heart defect and neonatal hepatitis in a female. Most remarkable was a clinically normal man who was the father of a mongol child. Fraccaro and co-workers[7] found that he was trisomic for chromosome no. 19.

Another case has been described by Hayward and Bower.[1] This was a boy of three years of age who was mentally retarded and suffered from frequent convulsions. A port-wine naevus covered the distribution of the first and second divisions of the right trigeminal nerve and there was buphthalmos of the right eye. X-ray of the skull revealed extensive calcification in the right parieto-occipital region. There were no features of mongolism — in fact the patient showed a completely characteristic Sturge-Weber syndrome. Bone marrow was cultured by the now standard method. In all the cells studied an extra small acrocentric chromosome was found which was identified as probably no. 22 (the smallest of all).

The mechanism of production of trisomy is chromosomal non-dysjunction, which may occur at mitosis or meiosis (reduction division). Mitotic non-dysjunction may occur during early cleavage in the developing embryo, or during pre-meiosis mitosis in the adult gonad. The first type should produce somatic autosomal mosaicism; the second aneuploid meiotic cells whose behaviour would parallel that of cells in which the non-dysjunction occurs during meiosis.

Non-dysjunction during meiosis may occur during the first or second division. Since the second division is essentially a haploid mitosis, the consequences of non-dysjunction at this phase are similar to those occuring during mitosis. Non-dysjunction at the first division may be produced by several different mechanisms. One of them is a failure of proper bivalent formation which may arise in three ways: (*a*) if the chromosome pair concerned are non-homologous, (*b*) from failure of pairing (asynapsis) between homologous chromosomes, and (*c*) from desynapsis (i.e. failure of chiasma formation after pairing). The first of these must depend entirely upon genetic properties.

Which of the various possible abnormal mechanisms accounts for non-dysjunction in any of the defects mentioned is quite unknown. Nor can we say for certain that a direct causal relation exists between chromosome aberration and clinical defect; the trisomy might merely be an accompanying phenotypic effect or, in some instances, a chance association. In mongolism, however, with a fairly uniformly abnormal chromosomal pattern, it is certainly tempting to believe that the autosomal trisomy is the cause of the abnormal phenotype.

1. Hayward, M. D. and Bower, B. D. (1960): Lancet, **2**, 844.
2. Böök, J. A., Fraccaro, M. and Lindsten, G. (1959): Acta Paediat., **48**, 453.
3. Jacobs, P. A., Baikie, A. G., Court-Brown, W. M. and Strong, J. A. (1959): Lancet, **1**, 710.
4. Lejeune, J., Gantier, M. and Turpin, R. (1959): C.R. Acad. Sci., **248**, 602.
5. Patau, K., Smith, D. W., Therman, E., Inhoorn, S. L. and Wagner, H. P. (1960): Lancet, **1**, 790.
6. Edwards, J. H., Harnden, D. G., Cameron, A. H., Crosse, V. M., Wolff, Frankhauser, G. and Humphrey, R. R. (1960): J. Exp. Zool., **115**, 207.
7. Fraccaro, M., Kaijser, K. and Lindsten, G. (1960): Lancet, **1**, 724.

* More have been described since this article was written.

AN OUTBREAK OF SUSPECTED CHIKUNGUNYA FEVER IN NORTHERN RHODESIA

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In 1957, Dr. James Gear, of the Poliomyelitis Research Foundation in Johannesburg, and Dr. F. P. Reid published an account[1] of an outbreak of dengue-like fever in a number of persons who had visisted the Kruger National Park in the Northern Transvaal. Their patients suffered from a febrile condition of sudden onset with headache, photophobia, pain in the joints and muscles, sore throat, occasional cough, and a maculo-erythematous rash appearing between the second and sixth days. Prolonged convalescence and recrudescence of pain were a feature of the disease. They regarded the disease as probably identical with Chikungunya fever, an outbreak of which had been described in Tanganyika in 1952 - 53.

In May 1959 an outbreak of a condition closely resembling that described by Gear and Reid occurred in Luanshya, Northern Rhodesia. It differed, however, in one important respect — all the Rhodesian cases had enlargement of the superficial lymph nodes, whereas this was not a feature of the Northern Transvaal outbreak. In addition, the last 3 cases of the Rhodesian series had definite pneumonic changes in the lungs.

CASE REPORTS

Early in May 1952 2 boys were admitted to the Roan Antelope Copper Mines Hospital at Luanshya with rigors, fever and vomiting. Both boys had been camping near Bancroft, on the Kafue River, for a week or more and remembered being badly bitten by mosquitoes. Malaria was excluded by repeated blood smears.

Case 1 (aged 16 years). This patient was admitted on 9 May 1959, having had rigors, fever, vomiting and diarrhoea for 5 days. The fever subsided temporarily, but a pink macular rash appeared on the chest, abdomen and arms on 11 May,

when the superficial lymph nodes in the posterior cervical, axillary, and inguinal regions became enlarged. The conjunctivae were injected. Further enlargement of these lymph nodes occurred on 13 May, and the spleen became palpable. Fever persisted until 18 May. When he was seen on 30 May the lymph nodes were unchanged, the spleen was no longer palpable and the patient stated that since leaving hospital he had had pain in the right knee and both ankles for several days.

Case 2 (aged 15 years) was admitted on 15 May 1959, having had rigors, bouts of sweating, fever, and vomiting for 2 days. On 19 May a profuse macular rash appeared on the trunk, and generalized lymph node enlargement was noted. The conjunctivae were deeply injected. The spleen was not enlarged. The next day the lymph nodes in the axillae and groins were enlarged to the size of marbles. When seen on 30 May the conjunctivae were still injected, and the lymph-node enlargement was unchanged. There had been pain in the ankles for several days.

Total white-cell counts in the first 2 cases were normal, with 7-8% and 10-17% monocytes respectively. Paul-Bunnell tests were negative in each case.

Two adults were admitted to the male ward of this hospital a few days after cases 1 and 2. The first had been at Bancroft 8 days before, and had been fishing elsewhere on the Kafue River frequently before and after that time. The other patient had been out fishing on the Kafue River both 8 days and 3 days before admission.

Case 3 (aged 39 years) was admitted on 22 May 1959, having had rigors, fever, pain and swelling of the right elbow, wrists, ankles and right knee, with stiffness of the right shoulder. Next day the superficial lymph nodes were enlarged above the left humeral epicondyle; also in the neck, axillae, and groins; and a sore tongue and throat developed. The convalescence was prolonged with weakness, lassitude, and stiffness in the right shoulder for 2 months. The white-cell count was within normal limits, with monocytes up to 11%, and the erythrocyte sedimentation rate was normal (Wintrobe 14 mm. and Westergren 12 mm. in the first hour).

Case 4 (aged 26 years) was admitted on 25 May 1959, having had high fever and pain in the back and legs for 1 day. During the next 2 days the conjunctivae became injected, and the superficial lymph nodes in the posterior cervical, axillary, and inguinal regions became enlarged. A faint pink erythematous rash appeared on the face and trunk, and there was soreness of the lips, tongue, and hard palate. The white-cell count was within normal limits. The lymphatic enlargement was unchanged when seen on 10 June. Muscular pain persisted for 3 months.

Case 5. I was now called to see a very important visitor to the Territory who, while staying in Luanshya, had become ill with slight pyrexia, adenitis and a slight erythematous flush. He said he had been 'eaten alive' by mosquitoes when fishing on the Luapula River 10 days before. He returned to Salisbury where a diagnosis of glandular fever was made by his physician.

On 3 and 4 June 1959 5 more cases occurred — none of these patients had been away from Luanshya, but 3 of them, R.K. a man of 37, M.K. his son aged 6 years, and A.J. a boy aged 6 years, lived in adjoining houses in a section of the residential area which had suffered a most unusual and unseasonable plague of *Culex fatigans* mosquitoes, the breeding places of which were found to be drums of liquid manure in various gardens in that neighbourhood.

The fourth patient, M.R., a girl of 12 years (the only female in the series) had been to an outdoor party, in the evening, in the garden of a house directly behind the 2 mentioned above, on the night of 22 May.

Three of the 4 patients complained of sore throat, 3 had a macular rash, 2 had pains in the joints at the time of onset, and all had generalized adenitis.

Case 10 (aged 43 years) was the last of the group of patients admitted on 3 and 4 June, and presented with swelling of the wrist- and ankle-joints and the smaller joints of the feet and hands. Two days later the superficial lymph nodes became enlarged. The white-cell count and the erythrocyte sedimentation rate were normal (18 mm. Wintrobe and 8 mm. Westergren). Pain in certain joints, the wrists especially, persisted for more than 3 months.

The last 3 cases in the group (11 - 13) differed in that they all developed pneumonic complications. They presented signs and symptoms similar to those recorded in the other cases, however, and I consider that they might have been affected by the same virus.

Case 11 (aged 5 years) was admitted on 11 June 1959. He looked severely ill, had rigors, fever and delirium at night and showed increasing abdominal distension and tenderness, with enlargement of the cervical lymph nodes only. Four days after admission radiological evidence of pneumonia was found although there were no physical signs in the chest. He had been given 'achromycin' the previous day, but this had had no effect on the pyrexia, and a course of chloramphenicol in full dosage was started on 20 June. The temperature fell to normal on the day the treatment started, but rose 3 days later and persisted until 5 July. The child remained weak and ate poorly for a further fortnight.

Case 12 (aged 38 years) was the father of the last case, and his illness started with a rigor and fever on 14 June 1959; next day the cervical, axillary, and inguinal lymph nodes were enlarged and a faint macular rash was present. The conjunctivae became injected. Fever had subsided by the third day, but on the fourth day of the illness the temperature rose again; backache, vomiting, deeply-injected conjunctivae and flushed skin developed, and later in the day a loud friction-rub was heard in the left axilla. X-rays showed a broad opaque band in the left lung, and the loud friction-rub persisted for a week. Lassitude and weakness prevented him from returning to work for 2 months in all. The Widal and Weil-Felix reactions were negative.

Case 13 (aged 20 months). The final patient in the series, a child of 20 months, had been regarded as suffering from measles on 24 June but, when admitted 9 days later, still had a heavy macular rash all over the trunk, enlarged superficial lymph nodes in the neck, armpits, and groin, and a pleuritic-friction rub and consolidation in the left lung, with a distended and tender abdomen. The illness responded slowly to a week's course of chloramphenicol and the patient recovered in 2 - 3 weeks.

FURTHER LABORATORY INVESTIGATIONS

Since the disease resembled both glandular fever and dengue fever in some respects, and a virus pneumonia was associated with several of the cases as well, I decided to ask Dr. James Gear, of the South African Institute for Medical Research, Johannesburg, whether he would be willing to investigate the condition in an attempt to indentify a virus as the causative agent. He kindly agreed to do this, and several blood and faecal specimens were sent to Johannesburg from convalescent cases. No virus was isolated, but the serum of one patient, V.R. (case 1), when tested against Semliki Forest and Chikungunya antigens, showed inhibition of the Semliki Forest antigen in a dilution of 1:40 and of Chikungunya antigen in a dilution of 1:640, no end-point being reached with this antigen. Complement fixation tests against these 2 antigens were also carried out. There was no fixation against Semliki Forest antigen, but complement was fixed to a dilution of 1:32 against Chikungunya. Later the serum

TABLE I. SIGNS AND SYMPTOMS OF 13 PATIENTS IN THE NORTHERN RHODESIAN OUTBREAK

Case	*Initials*	*Age in years*	*Adenitis at onset*	*Arthritis at onset*	*Arthritis in convalescence*	*Sore throat*	*Sore tongue*	*Rash*	*Severe muscular pain*	*Conjunctivitis*	*Splenic enlargement*	*Pneumonia*	*Prolonged convalescence*
1	V.R.	16	+		+			++		+	+		
2	G.v.B.	15	++		+			++		+			
3	W.A.J.	39	+	++		+	+						+
4	R.H.	26	+			+	+	+	+	+			
6	R.K.	37	+	+				+		+			
7	M.K.	6	+	+		+							
8	A.J.	6	+			+		++		+			
9	M.R.	12	+			+		++		+			
10	D.A.O.	43	+	++									
11	J.D.	5	+									+	+
12	R.J.D.	39	+					+	+	+		+	+
13	N.J.	1½	+					++				+	
Total no. of patients showing each sign and symptom			12	4	2	5	2	8	2	7	1	3	3

+ = present to a moderate degree; ++ = present to a severe degree. Case 5 is not included in the above table.

of G.v.B. (case 2) was reported by Dr. R. H. Kokernot, of the Rockefeller Foundation, Johannesburg, to inhibit the agglutination of Chikungunya antigen in a dilution of 1:640 or greater, while Semliki Forest and Uganda antigens were inhibited in dilutions of 1:40.

In view of these findings Dr. Gear wrote that it would be worth while to test sera from patients who had recovered. Accordingly sera from 7 more patients were sent for testing and in 3 of these cases (3, 6 and 7) the Chikungunya antigen was inhibited in a dilution of 1:320.

DISCUSSION

There seem reasonable grounds for the assumption that most of these cases represent one clinical entity, and their general resemblance to the cases of Gear and Reid[1] is striking.

They showed the same sharp pyrexial bout of sudden onset and fairly short duration, while some had sore throat, an erythematous rash (which was usually macular), injection of the conjunctivae, muscular pains and involvement of several joints (Table I).

A particularly noticeable point of resemblance was the manner in which the joint pains tended to persist in spite of normal erythrocyte sedimentation rates, and the prolonged period of convalescence. On the other hand, it is certainly surprising that only in one instance was lymph gland enlargement noted in the Transvaal cases, while it was a constant and prominent feature in our series. However, I think the serological findings, incomplete and inconclusive though they undoubtedly are, do suggest that the Chikungunya virus may be implicated in the Luanshya cases.

The question of the disease vector is interesting. I have been informed by Dr. Gear in a private communication that he considers the vector in his series was probably a mosquito, though this could not be proved.

I have already stated that the sudden appearance of very large numbers of *Culex fatigans* mosquitoes in Luanshya in May and early June was quite exceptional; it caused numerous bitter complaints from residents in a small locality in Luanshya where 5 cases probably originated, and it is reasonable to assume that a number of the earlier group of cases had also been bitten by this mosquito. Although it is obviously impossible to draw definite conclusions, *Culex fatigans* must, for the time being, be regarded as a definite suspect when an attempt is made to track down the propagator of this disease.

SUMMARY

A series of 13 cases of Chikungunya fever occurring in Luanshya, Northern Rhodesia, during May and June 1959, is reported. Three of the cases had signs of pneumonia. Laboratory investigations, which helped to identify a virus as the causal agent, are described. The similarity between the cases reported and those which occurred in an earlier outbreak in the Transvaal, are noted. The vector of the disease is suspected to be the mosquito *Culex fatigans*.

I am greatly indebted to Dr. James Gear, Director of the South African Institute for Medical Research, Johannesburg, for his assistance and advice. I also wish to thank Dr. R. H. Kokernot of the Rockefeller Foundation, Johannesburg, for his kindness in undertaking the investigation of these cases.

REFERENCE

1. Gear, J. H. S. and Reid, F. P. (1957): S. Afr. Med. J., 31, 253.

A STUDY OF ATTITUDES AND KNOWLEDGE CONCERNING OBESITY IN AN URBAN AFRICAN COMMUNITY

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PURPOSE OF THIS STUDY

Recent work[1] has shown that 68·9% of a sample of urban African women weighed 10% or more in excess of American women of the same age and height. If health education to counteract obesity is to be successful, it must be based on some understanding of the existent attitudes and knowledge concerning obesity. This study was undertaken for 2 purposes — firstly to ascertain the beliefs and understanding of urban Africans about obesity, and secondly to see whether these are affected by age, sex, marital status, ethnic group or educational standard.

MATERIAL AND METHODS

Data were obtained by a questionnaire administered by a health educator in Lamontville — an urban municipal housing scheme for Africans. A series of 63 homes from a 1 in 65 sample was randomly selected and all volunteers over 16 years of age in the home were interviewed. Of the individuals in the sample 63% completed questionnaires.

The 112 respondents included 34 men and 78 women, 87 married and 22 single people, 81 Zulus and 28 non-Zulus. Of the respondents, 44 were less than 30 years of age and 29 were between 30 and 40 years; 31 had less than 5 years of schooling, 47 had between 5 and 8 years and 33 had 9 years or more. Discrepancies in the totals are due to incomplete returns. The smaller proportion of men than women in the sample is due to the practical difficulties of contacting working men at their homes.

A 'majority' opinion and a 'strongly-held' opinion are taken as those held by more than 50% and 75% respectively of the respondents and they will be indicated in Tables I - VI by horizontal lines dividing off those with a 75% or more, 50% or more and under 50% agreement.

The minimum number of respondents answering any item was high, viz. 103 (97·2%). The answers were expressed as 'True/false/don't know'. Those statements in which the proportion of 'don't knows' is high are indicated in the tables.

Levels of significance were based on the chi-square test (using Yates' correction).

FINDINGS

Table I lists the statements related to causes of obesity and shows that there is strongly held agreement that obesity is common in urban African women. The majority assert that obesity is associated with wealth, happiness and age and not with worries. Just over half the respondents agree that excess maize, fat or any kind of food will cause obesity. Opinion is equally divided about the effects of inheritance, childbearing, dietary intake in excess of the body's requirements and lack of exercise. A minority of respondents say that too much sugar causes obesity. This would appear to be at variance with the previous view that excess of any kind of food will cause obesity.

TABLE I. CAUSES OF OBESITY

Statement	% *Agreement*
Fatness is commoner:	
—in women than men	96·3
—in towns than in the country	86·5
—in Africans than Europeans	76·14
Fatness is caused by:	
—riches	72·9
—happiness	65·5
Fatness is not due to worries	63·9
Age causes fatness	63·6
Too many vegetables can cause fatness	59·4
Too much sugar can cause fatness	41·8 (17·3% don't know)
Eating too much of any kind of food will make you fat	56·0
Fatness can be caused by sickness	55·8
Too much maize products can cause fatness	55·0
Too much fat can cause fatness	52·7
Eating more than your body needs causes fatness	50·4
Childbearing causes fatness	50·9 (19·4% don't know)
Fatness is inherited	50·9 (19·4% don't know)
Fatness is due to lack of exercise and laziness	47·7

Table II lists the statements on food, eating habits and obesity and indicates strongly-held opinions that the diet of Africans contains excess maize and sugar products, that diet and health are related, that there is variation in the response to similar diets and that individual likes are the

TABLE II. DIET AND OBESITY

Statement	% *Agreement*
Africans eat more maize products than they need	91·0
On the same diet one person can be thin, another fat	80·2
Africans eat more sugar and sweet foods than they need	79·5
Your health is affected by your food	78·7
People should eat whatever kind of food they like	78·3
Africans eat more fat than they need	74·8
Too much food affects your health	73·8
Whenever you are hungry you should eat	68·8
Eating does not help you forget worries	67·0
People should eat as much as they like	66·1
A man should not get the biggest share of the food	63·4
Too little food affects your health	61·7
You should only eat when you have an appetite	60·2
Adults should eat more than children	57·3
Men should not eat more than women	46·8
Boys should eat more than girls	40·5 (17·4% don't know)

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criterion for choice of diet. The majority agree that excess or deficiency of food can affect health, that appetite is a criterion for the time and quantity of meals and that men should not get the biggest share of the food. Opinion is divided whether men and women or boys and girls should eat the same quantities of food.

Table III lists the statements related to obesity and the stages of development of an individual and shows that the majority regard thinness in children and babies as not necessarily abnormal. Opinion is divided on whether

TABLE III. OBESITY AND DEVELOPMENT

Statement	% *Agreement*
A thin child is not a sick child	82·1
A thin baby is not a sick baby	64·8
A pregnant woman can be too fat	55·0
A baby can be too fat	54·5 (20·7% don't know)
A child can never be too fat	48·6
It is good for a woman to be fat when pregnant	48·6
Adolescent girls should get fat	48·1

adolescent and pregnant women should be fat and whether there is an upper limit of what can be regarded as normal fatness in babies and children.

Table IV lists the statements related to the results of obesity and indicates that there is strongly-held agreement

TABLE IV. EFFECTS OF OBESITY

Statement	% *Agreement*
Fat people become short of breath and suffer from joint pains	91·9
Fatness affects the heart	89·2
It is worse for a fat person to get sick	86·1
Thin people live longer than fat people	63·4 (21·4% don't know)
Diabetes is common in fat people	61·1 (26·9% don't know)
Fat women conceive less easily than thin women	57·7 (33·3% don't know)

about the harmful effects of obesity. The majority think that diabetes and infertility may be associated with obesity, and longevity with thinness.

Table V lists those items related to attitudes to obesity and shows strongly-held opinions that obesity is undesirable, both from the health and aesthetic points of view, but opinion appears to be equally divided whether or not

TABLE V. ATTITUDES TO OBESITY

Statement	% *Agreement*
It is not healthy to be too fat	91·0
Fatness is worse than thinness	77·5
Fat people are not attractive	75·0
A fat man is not better than a thin man	68·5
It is a sign of health to be fat	48·1

fatness is a sign of good health. The majority (68·7%) do not want to change their weight in either direction.

Table VI lists items related to weight reduction and shows that the majority agree that fat people should reduce

TABLE VI. WEIGHT REDUCTION

Statement	% *Agreement*
The best person to help in reducing weight is the doctor or nurse	95·4
An *inyanga* (witchdoctor) is not the best person to help in reducing weight	90·8
To reduce weight you must exercise more	84·9
A fat person should reduce in weight	74·5
To reduce you must eat less	68·2 (19·1% don't know)

by consulting medical personnel, increasing their exercise and decreasing their dietary intake.

Table VII shows that there are a few significant differences between men and women, single and married persons, and poorly and highly educated individuals regarding some of the causes of obesity, dietary requirements and normal weight standards.

DISCUSSION

It has been shown that death rates are higher for obese persons than for those of normal weight or below.[2] In communities in which obesity is a clinical problem it is therefore important that weight control or reduction should be acceptable.

Every cultural group has its own set of attitudes, beliefs and knowledge concerning diet and health. These will directly affect their response to attempts at weight control or reduction. In addition, eating habits and the criteria of the food requirements of the two sexes and of individuals at different stages of development are also culturally influenced.

Bryant[3] in his description of the Zulu people as they were before the White man came states that men 'with corporations' and women with 'substantial buttocks' were especially admired. It is possible that these views may be undergoing modification as a result of urbanization and industrialization and diffusion of new concepts of health and disease. This would appear to be so from the fact that the majority of the respondents agree that thin, rather than fat, people are attractive, that it is not healthy to be too fat, that fatness is worse than thinness, and that it is worse for a fat person to get sick. The association that is accepted by the majority between fatness on the one hand and riches and happiness on the other may possibly mean that obesity is favourably regarded as a visible manifestation of material and emotional well-being.

The community in this study has received health education as a part of the comprehensive medical and health care programme of the Institute of Family and Community Health of the University of Natal.[4] * It is possible that consequently their knowledge concerning obesity is slightly more advanced and sophisticated than that of other urban African communities who have not yet received this kind of health education.

Briefly, the strongly held views may be summarized as follows: Diet and health are related, obesity produces

* This Institute closed down on 31 January 1961, after this article was written.

TABLE VII. SIGNIFICANT OPINION DIFFERENCES

Statement	Associated with	Respondents	Agreement No.	Agreement %	Level of significance of differences
	(a) Sex				
Lactation causes obesity	Men	34	6	17·6	P<·01
	Women	76	34	44·7	
Lactating women should be fat	Men	34	13	38·2	P<·02
	Women	67	45	67·2	
	(b) Marital status				
Childbearing causes obesity	Single	18	7	47·0	P<·05
	Married	67	46	68·6	
Boys should eat more than girls	Single	17	3	17·6	P<·01
	Married	73	41	56·2	
Men should eat more than women	Single	15	2	13·3	P<·02
	Married	76	39	51·3	
	(c) Education				
Sign of health to be fat	Less than 5 years	28	19	67·8	P<·05
	5 years and more	74	31	41·9	
A baby can never be too fat	Less than 5 years	32	21	65·6	P<·01
	5 years and more	77	27	35·1	
Adolescent girls should be fat	Less than 9 years	71	45	63·4	P<·01
	9 years and more	25	6	24·0	
	(d) Ethnic group				
Obesity caused by eating too much fat	Zulu	70	48	68·6	P<·05
	Non-Zulu	21	8	38·1	

harmful effects, fatness is not attractive, the diet of Africans contains excessive maize and sugar products, exercise is necessary for weight reduction, and people should eat whatever kind of food they like. This final statement that an individual's likes should be the criteria for choice of diet might explain some of the difficulties experienced in modifying the eating patterns of obese patients.

It is necessary to point out that this study did not define obesity and that the criteria of what constitutes obesity still have to be determined in this community. It is possible that what is regarded as the upper limit of normal weight may be very much higher than clinically established norms.

It would appear that the factors of sex, age, marital status and educational standard do not influence to any great extent attitudes, beliefs and knowledge about obesity.

SOME PRACTICAL IMPLICATIONS

There would appear to be acceptance of health promotive attitudes and knowledge about obesity in the urban African community which was studied. The aspects which seem to require further health education are the relationship between dietary excess and obesity, the maximal limits of normal weight for the individual at different developmental stages, e.g. infancy, adolescence, and pregnancy. The need for more knowledge on methods of weight control and reduction is indicated. Finally it appears that there is no necessity for specific programmes for the different sexes, age groups or educational levels in the community.

SUMMARY

The findings of a study of attitudes, beliefs and knowledge of urban Africans about obesity are presented and some of the practical implications discussed.

Thanks are due to staff members of the Institute of Family and Community Health, Durban, viz. Mrs. G. Ntwasa for completing the questionnaires and Messrs. W. H. Pietersen and S. J. Maharaj for assistance with the extraction of the results. A copy of the questionnaire can be obtained from the author.

REFERENCES

1. Slome, C., Gampel, B., Abramson, J. H. and Scotch, N. (1960): S. Afr. Med. J., **34**, 505.
2. Marks, H. H. (1957): Metabolism 6 : 417 (Sept.).
3. Bryant, A. T. (1949): *The Zulu People as they were before the White Man Came*. Pietermaritzburg: Shuter and Shooter.
4. Kark, S. L. and Steuart, G. W. (1957): Hlth Educ. J., **15**, 131.

HERPES SIMPLEX STOMATITIS IN CHILDREN: ITS CLINICAL PICTURE AND COMPLICATIONS AS SEEN IN CAPE TOWN*

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Stomatitis is defined for the purposes of this paper as an infection that involves the tongue, the gums, and the mucous membrane of the hard palate, cheeks and lips. Angular stomatitis and cheilosis from deficiency diseases are not included under this heading.

The various types of stomatitis seen in children include: (1) herpes simplex stomatitis; (2) thrush or monilial stomatitis; (3) Vincent's stomatitis; (4) catarrhal stomatitis due to systemic disease, drugs or trauma; and (5) noma or acute gangrenous stomatitis of the mouth due to pyogenic organisms in severely debilitated children.

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PICTURE OF HERPES STOMATITIS

Clinical Description

Ulcero-gingivo-stomatitis caused by the herpes simplex virus is probably the commonest cause of stomatitis in childhood.[1,2] Clinically it has the following characteristics: The age incidence is between 1 and 6 years. The symptoms are mainly those of refusal to eat because of painful mouth. Along with this there is general malaise and fever. The children are extremely restless and unhappy. On examination, small

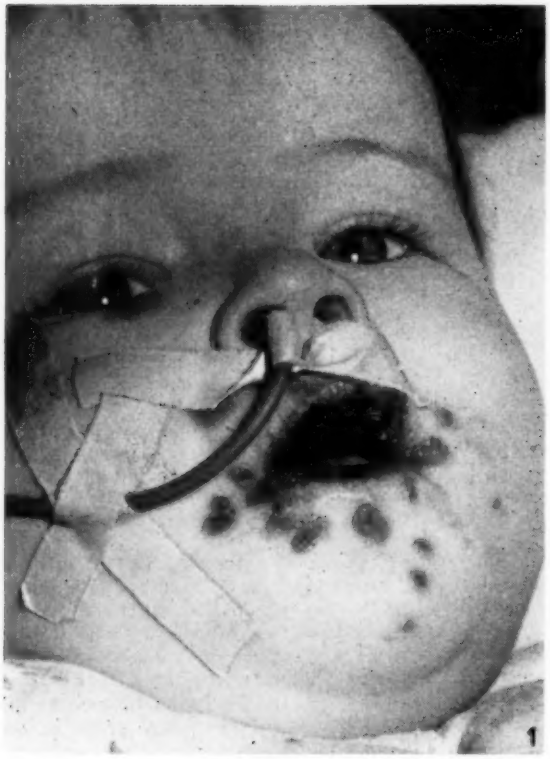

Fig. 1

herpetic lesions are frequently present on the lips and face (Fig. 1). Inside the mouth the initial lesion is a vesicle which is seldom seen because it ruptures early. The ulcer that results is quickly covered by a greyish-yellow membrane and this frequently resembles a plaque. The lesions can be found on the tongue, the palate, and the mucous membrane of the cheek and lips. They vary in size but are usually between 0·3 and 1 cm. in diameter. The gums are swollen and red and extend down between the teeth. They bleed easily on being touched by a spatula. Frequently there is a submental or submandibular adenopathy.

Clinical Course

This lasts from 10 to 16 days after the onset of symptoms and no treatment is at present known that will shorten this course. During this time the mouth continues to be extremely painful and usually there is a persistent pyrexia which may rise to 105°F. The severity of the systemic reaction thus sometimes seems to be out of proportion to the local lesions in the oral cavity. On recovery there is complete resolution of the lesions without scarring.

Epidemiology[*]

At birth, infants have a passive immunity to herpes simplex infection. This immunity disappears by the age of 6 months, after which the infants become susceptible to primary infection with the virus. It is generally believed that about 90% of primary herpetic infections are sub-clinical, while in the other 10% of infections there is a manifest clinical state in the form of stomatitis which occurs most frequently between the ages of 1 and 3 years. A non-clinical carrier state exists as well and is more common in children from 7 months to 2 years of age (20%) than in adults (2·5 %). For this reason patients with clinical herpes usually give no history of contact with herpes infections.

Following the primary invasion of the virus both in clinical and subclinical infections there is a rise of antibody titre in the serum. The virus, however, remains established in the host but becomes latent. In certain susceptible individuals there are recurrent attacks of herpes simplex stomatitis. These attacks occur at times of stress such as generalized systemic disease, emotional upsets, menstruation, sunburn, etc. The ulcero-gingivo-stomatitis infection seen in children usually represents the primary infection with herpes simplex virus.

Differential Diagnosis

1. *Thrush.* This occurs in particular in children under the age of 1 year. It is characterized by small milky-white lesions on the tongue and the oral mucous membrane. The mouth looks red and inflamed but there is no true ulceration and the gums are not affected as in herpetic stomatitis. There is as a rule no adenopathy or lesions on the face. Thrush responds rapidly to therapy with 1% gentian violet or 'mycostatin'.

2. *Vincent's stomatitis.* This is more difficult to differentiate since so many cases of herpetic stomatitis have secondary infection with Vincent's organisms. However, Vincent's stomatitis is rarer in children than in adults. It is characterized by severe halitosis, and the gums tend to be retracted rather than hypertrophied round the bases of the teeth. Treatment with penicillin will cause resolution of the symptoms within a couple of days.

3. *Catarrhal stomatitis* due to systemic disease, drugs or trauma must be differentiated on the history.

4. *Noma or gangrene of the mouth* is a gross condition. It is very rarely seen in this country.

HERPES SIMPLEX STOMATITIS IN CAPE TOWN

During the last 4 years, 121 cases of herpes simplex stomatitis have been seen in the wards of the Red Cross War Memorial Children's Hospital and the paediatric wards of Groote Schuur Hospital. An analysis of these cases is presented in Table I. It will be noted that the mean age was 20 months, which

TABLE I. ANALYSIS OF CASES

Hospital	*Number of cases*	*Mean age*	*Male*	*Female*	*European*	*Non-European*	*Mal-nourished*	*Deaths*
Red Cross Hospital	81	20 ± 13 months	44	37	16	65	55 (E=4)	26
Groote Schuur Hospital	40	20 ± 13 months (excluding 3 European children aged 7, 8 and 10 years respectively)	21	19	14	26	24 (E=2)	7
Total	121		65	56	30	91 (75%)	79 (65%)	33 (27%)

E=European.

TABLE II. ANALYSIS OF DEATHS

Hospital	*No.*	*European*	*Mal-nourished*	*Confirmed by autopsy*	*Autopsy negative*	*No autopsy*
Red Cross Hospital	26	1	25	13	5	8
Groot Schuur Hospital	7	0	6	3	1	3
Total	33	1	31 (94%)	16 (49%)	6 (18%)	11 (3%)

agrees well with what is described in the literature. The sex incidence showed an equal distribution between males and females. Of the cases 75% were non-European. The majority of the children (65%) were malnourished, i.e. they conformed to the clinical diagnosis of kwashiorkor or marasmus, or weighed less than 65% of the expected weight for their age. The death rate was 27% which was surprisingly high. An analysis of the deaths is shown in Table II. From this it can be seen that all except 2 deaths occurred in grossly malnourished children. Even the one European child who died suffered from protein malnutrition. In almost half (49%) of the 33 deaths there was autopsy evidence of generalized herpes simplex infection.[4] In these children there was a high incidence of coma or convulsions, hepatomegaly, and bleeding tendency or purpura (Table III). In cases that recovered a bleeding tendency was noticed in only 1 and convulsions in 3.

TABLE III. CLINICAL FEATURES OF PATIENTS WHO DIED

Cases	*Age range*	*Coma or convulsions only*	*Bleeding tendency or purpura*	*Convulsions and bleeding tendency*	*Large liver*
16 proved cases	9 - 24 months	11 (69%)	7 (44%)	4 (25%)	12 (75%)
17 unproved cases	11 - 30 months	3 (17%)	5 (29%)	0	8 (47%)
Total 33		14 (42%)	12 (36%)	4 (12%)	20 (60%)

Treatment

All patients received antibiotics and supportive therapy with intravenous fluids where indicated. Gamma globulin in large doses was given to 17 severely-ill children, of whom 9 died. It is therefore not possible to say whether it had any beneficial effect. It is probable that the gamma globulin was given too late, i.e. after generalized dissemination of the virus had taken place. In view of the general state of shock and collapse that precedes death and the frequent occurrence of adrenal necrosis[4] it was thought that cortisone might be beneficial. It was given to 7 patients where generalized spread of the virus was suspected clinically. Of these, 6 died.

DISCUSSION

The clinical features of the series of cases of herpes simplex stomatitis reported in this paper conformed in most respects to what is well described in the paediatric literature. This applies in particular to the age incidence, 1 - 3 years, and the preponderance of infections among the lower socio-economic groups. In a study of the Pretoria Bantu population Coetzee[5] found that, at birth, high antibody levels (passively transferred from the mother) were present in 100% of cases studied. Between the ages of 3 months and 5 years there were a variable number of negative sera but at 6 years 100% of the population again had high antibody levels. Universal primary infection in Pretoria therefore occurred before the age of 6 years. It would seem that the pattern is similar in Cape Town and would account for our seeing so few primary infections among lower socio-economic groups after that time. The 3 older children with herpes stomatitis in our series were all Europeans of higher economic status.

Generalized spread of the herpes virus with a fatal outcome has been described by several authors. It sometimes occurs in children with eczema and is known as Kaposi's varicelliform eruption. All reported deaths have been in the neonatal period apart from this type and 2 cases reported by Zuelzer and Stulberg[6] in 1952. In the series reported in this paper, it is of interest that there were 16 cases aged 9 - 24 months in which generalized spread of herpes virus was proved at autopsy.† The cause of this high incidence of fatal generalized herpes simplex infection in Cape Town is obscure. It is tempting to suggest that severe malnutrition, which was an almost universal association with these fatal cases, favours the generalized spread of the virus.

SUMMARY

Stomatitis resulting from herpes simplex virus infection is the commonest form of stomatitis in childhood between the ages of 1 and 6 years. Symptoms are sometimes severe enough to warrant hospitalization. A series of 121 cases at 2 hospitals in the Cape Peninsula has been analysed. Thirty-three of these cases died and, in 16 of these, generalized herpes simplex infection was found at autopsy. The relationship of severe malnutrition in the form of kwashiorkor or marasmus to generalized spread of herpes simplex virus with a fatal outcome is discussed.

Thanks are due to Mr. B. Todt for the photograph and to Dr. J. G. Burger, Superintendent of Groote Schuur Hospital, and Dr. J. F. W. Mostert, Superintendent of the Red Cross War Memorial Children's Hospital, for permission to publish these cases.

REFERENCES

1. Nelson, W. E. (1959): *Textbook of Pediatrics*, 7th ed. Philadelphia: Saunders.
2. McNair Scott, T. F. (1955): G.P., **12**, 59.
3. *Idem* (1957): Amer. J. Opthal., **43**, part 2, 134.
4. McKenzie, D., Hansen, J. D. L. and Becker, W. (1959): Arch. Dis. Childh., **34**, 250.
5. Coetzee, J. H. (1955): S. Afr. J. Lab. Clin. Med., **1**, 52.
6. Zuelzer, W. W. and Stulberg, C. S. (1952): A.M.A. Amer. J. Dis. Child., **83**, 421.

† See article by Dr. D. McKenzie on p. 133 of this issue.

DISSEMINATED HERPES SIMPLEX INFECTION*

D. McKENZIE, M.MED. (PATH.), *Red Cross War Memorial Children's Hospital, Rondebosch, Cape, and Department of Pathology, University of Cape Town*

Infection with herpes simplex virus usually manifests clinically as vesicular lesions localized to the epithelial surfaces of the lips, face and mouth and, less frequently, the genitalia.

Dissemination in internal organs has been described by several authors (Table I).[1-11]

AUTOPSY FINDINGS IN HERPES SIMPLEX

Sixteen cases of disseminated herpes simplex virus infection have been seen at autopsy in Cape Town over a period of 18 months. These are summarized in Table II. Eight of these cases, together with the morbid anatomy and histology of the lesions, have been reported elsewhere.[12]

None of these cases were in newborn infants. Fifteen of them were from 9 to 34 months of age and 1 was 2½ months old. All save 1 were obviously malnourished. All these deaths occurred in non-European children. In 10 cases the liver lesion was macroscopic and in 4 microscopic, while in 2 cases no liver lesions were seen. The adrenal lesions, where present, were microscopic. In 1 case lung lesions due to herpes simplex virus were found. Other factors which possibly contributed to death are listed in the last column of Table II.

Oral Lesions

Lesions in the mouth and tongue are often masked clinically by becoming confluent and very extensive. The lesions are basically superficial ulcers with nuclear inclusions in the epithelial cells at the margins of the ulcers. Multinucleate giant cells, often with intra-nuclear inclusions, are not infrequently shed into the necrotic debris in the area of the ulcer. These giant cells are most characteristic and are found in only 3 conditions, viz. herpes simplex infection, herpes zoster and varicella.

The lesions frequently extend to involve the pharynx and oesophagus, and, while generally ulcerative in type, are occasionally hypertrophic, presenting as yellow nodules which

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September - October 1959.

consist histologically of a coagulum of nuclear debris and fibrin.

Liver Lesions

The oral lesions may well be the result of spread by continuity, but the macroscopic hallmark of disseminated herpes simplex infection is the liver lesion (Fig. 1). The lesion in the liver, consisting of small white foci of necrosis with a haemorrhagic halo, is pathognomonic and is seen in no other condition of which we are aware. The foci may vary in size, presumably with age.

Histologically the necrotic foci are infarct-like in character in that both parenchyma and stroma are equally destroyed. Usually only nuclear remnants are visible in the centre together with red cells, lying free in an area of necrosis, or, in older lesions, in a lake of haemolysed red cells. These areas merge abruptly into surviving liver parenchyma where the only abnormalities are in the cell nuclei. There is no evidence of inflammatory reaction or proliferation of liver cells but there is some dilatation of sinusoids around the lesion.

Nuclear inclusions are prominent in the liver and take on different forms. It is suggested that these may only be stages of development of the virus within the cell nucleus. The inclusions are either single uniform eosinophilic bodies filling the distorted and swollen nucleus, or multiple small eosinophilic bodies. At some stages, under oil immersion magnification, these eosinophilic bodies appear to be set in a basophilic matrix. Sometimes a clear halo separates the inclusion from the disintegrated chromatin network which has become marginated on the inner aspect of the nuclear membrane; this being frequently distorted.

Lesions in Other Organs

In the adrenal glands foci of necrosis, basically similar to those in the liver, are seen in both cortex and medulla. In none of our cases have these lesions been macroscopic. Nuclear inclusions are present but not with the frequency or variation in form seen in the liver.

In one case lesions, which it is believed can be attributed to the herpes simplex virus, were seen in the epithelial cells lining the bronchi and trachea. The same basic pattern of necrosis was evident. In one case also there was an encephalitis. Lesions in other organs were not seen in our cases.

Virus Studies

Virus studies were carried out on organs removed at autopsy in 6 cases (Table III). In case 16 the virus was isolated from the blood taken before death. This represents one of the few instances where the virus of herpes simplex has been isolated from the blood stream. Virus isolations were carried out by Dr. W. Becker in the CSIR and UCT Virus Research Unit of the Department of Pathology at the University of Cape Town.

DISCUSSION

No concrete explanation can be given for the frequently occurring cases of disseminated herpes simplex virus infection in our autopsy material. Nearly all the cases seen have been in severely malnourished children and it may well be that there is either lack of some vital protective mechanism in these cases, which permits of dissemination of the virus, or the altered cellular metabolism in these cases may render the cells of internal organs more accessible to circulating virus. Much investigation remains to be done on this score, but in conclusion attention should be focused on the macroscopic liver lesion, which is the morbid anatomical hallmark of disseminated herpes simplex virus infection.

TABLE I. DISSEMINATED HERPES SIMPLEX—AUTOPSY FINDINGS IN VARIOUS SERIES

Author	*Lesions*	*No. of cases*	*Virus isolated*
Hass[1]	Lesions in adrenal and liver, herpetic origin mooted	1 neonate	No
Wildi[3]	Encephalitis	1 neonate	Keratitis in rabbits
Smith *et al.*[8]	Encephalitis	1 neonate	Yes
Florman and Mindlin[4]	Encephalitis	1 neonate	Yes
Quilligan and Wilson[5]	Hepatic necrosis	1 neonate	Yes
Zuelzer and Stulberg[6]	Hepatic necrosis	8 cases (6 neonates, 2 older)	In one case
France and Wilmers[7]	Hepatic necrosis	2 neonates	No
Pugh *et al.*[8]	Hepatic necrosis	1 neonate	Yes
Williams and Jack[9]	Hepatic necrosis	2 neonates	In one case
Brain *et al.*[10]	Hepatic necrosis	1 neonate	Yes
Zuelzer and Stulberg[6]	Adrenal necrosis	Some of 8 cases	In one case
Williams and Jack[9]	Adrenal necrosis	2 neonates	In one case
Brain *et al.*[10]	Adrenal necrosis	1 older child with infantile eczema	Yes

Subsequent to the presentation of this paper a further neonate case was described by Bird and Gardener.[11] There were liver and adrenal lesions and the virus of herpes simplex was isolated from organs at autopsy.

TABLE II. AUTOPSY FINDINGS IN 16 CASES OF DISSEMINATED HERPES SIMPLEX

Case	*Race and sex*	*Age*	*Hepatic necrosis*	*Adrenal necrosis*	*Nutritional state*	*Other findings*
1	C.M.	12 mths	Microscopic	Microscopic	Kwashiorkor	Small intra-alveolar haemorrhage
2	C.M.	11 mths	—	Microscopic	Kwashiorkor	Intra-alveolar haemorrhage in lung and small abscesses
3	C.M.	16 mths	Macroscopic	Microscopic	Kwashiorkor	—
4	C.F.	12 mths	Macroscopic	—	Marasmus	Broncho-pneumonia and fibrinous pleurisy
5	C.F.	12 mths	Macroscopic	Microscopic	Kwashiorkor	Hyperosmolaric
6	C.M.	34 mths	Macroscopic	Microscopic	Marasmus	Bronchopneumonia
7	N.M.	15 mths	Macroscopic	Microscopic	Marasmus	Bronchiolitis
8	C.M.	9 mths	Microscopic	Microscopic	Kwashiorkor	Encephalitis
9	C.M.	15 mths	Microscopic	Microscopic	Normal—slight fatty liver	—
10	C.F.	12 mths	Macroscopic	—	Kwashiorkor	—
11	C.F.	2½ mths	Microscopic	—	Marasmus	Herpetic lung lesions. Hyperosmolaric with sagittal sinus thrombosis
12	C.M.	25 mths	Macroscopic	Microscopic	Marasmus	—
13	C.M.	15 mths	—	Microscopic	Kwashiorkor	Bronchiolitis and bronchopneumonia
14	C.F.	10 mths	Macroscopic	Microscopic	Marasmus	—
15	N.M.	14 mths	Macroscopic	Microscopic	Malnourished	Pneumococcal meningitis
16	C.M.	18 mths	Macroscopic	Microscopic	Kwashiorkor	—

C=Coloured, N=Native, M=male, F=female.

1

Fig. 1. Macroscopic liver lesions in disseminated herpes simplex.

SUMMARY

Autopsy findings in 16 cases of disseminated herpes simplex infection are listed. Virus studies were carried out on 6 of these cases. Attention is drawn to the striking macroscopic appearance of the liver.

TABLE III. VIRUS ISOLATION

Case No.	*Specimen examined*	*Virus isolation*
7	Liver	Positive.
10	Liver	Positive.
	Spleen	Positive.
	Lung	Positive.
	Heart, tongue, kidney, adrenal, brain and blood	Negative.
12	Liver	Positive.
	Spleen	Positive.
	Lung	Positive.
	Adrenal	Positive.
	Large bowel mucosa.	Positive.
	Tongue mucosa	Positive.
	Blood, heart, brain and kidney	Negative.
14	Liver	Positive.
15	Liver	Positive.
	Spleen	Positive.
16	Liver	Positive.
	Spleen	Positive.
	Lung	Positive.
	Adrenal	Positive.
	Tongue mucosa	Positive.
	Small bowel mucosa.	Positive.
	Oesophageal mucosa	Positive.
	Brain	Positive.
	Myocardium	Positive.
	Kidney	Positive.
	Blood	Positive.

Thanks are due to Mr. B. Todt for the photograph and to Dr. J. W. Mostert, Superintendent of the Red Cross War Memorial Children's Hospital, for permission to publish these cases.

REFERENCES

1. Hass, G. M. (1953): Amer. J. Path., **11**, 127.
2. Wildi, E. (1951): Rev. Neurol., **84**, 201.
3. Smith, M. G., Lennette, E. H. and Reames, H. R. (1941): Amer. J. Path., **17**, 55.
4. Florman, A. L. and Mindlin, A. L. (1952): A.M.A. Amer. J. Dis. Child., **83**, 481.
5. Quilligan, J. J. and Wilson, J. L. (1951): J. Lab. Clin. Med., **38**, 742.
6. Zuelzer, W. W. and Stulberg, C. S. (1952): A.M.A. Amer. J. Dis. Child., **83**, 421.
7. France, N. E. and Wilmers, M. J. (1953): Lancet, **1**, 1181.
8. Pugh, R. C. B., Newns, G. H. and Dudgeon, J. A. (1954): Arch. Dis. Childh., **29**, 60.
9. Williams, A. and Jack, I. (1955): Med. J. Aust., **1**, 392.
10. Brain, R. T., Pugh, R. C. B. and Dudgeon, J. A. (1957): Arch. Dis. Childh., **32**, 120.
11. Bird, T. and Gardner, P. S. (1959): Brit. Med. J., **2**, 993.
12. McKenzie, D., Hansen, J. D. L. and Becker, W. (1959): Arch. Dis. Childh., **34**, 250.

43ste MEDIESE KONGRES (M.V.S.A.), KAAPSTAD, 24 - 30 SEPTEMBER 1961 : 43rd MEDICAL CONGRESS (M.A.S.A.), CAPE TOWN, 24 - 30 SEPTEMBER 1961

RULES GOVERNING THE PRIZE FOR REGISTRARS IN ANAESTHETICS

Dr. R. A. Moore Dyke, of Port Elizabeth, has offered a prize for the best paper presented by a registrar in anaesthesia at the 43rd South African Medical Congress. The prize will consist of an illuminated scroll and books to the value of £25.

Entrants must comply with the following rules:

1. They must be full-time members of an anaesthetic department in a South African hospital, and the holder of a registrar or clinical assistant post in that hospital, and shall not be registered as a specialist in anaesthesia by the South African Medical and Dental Council.

2. The paper presented must be concerned with original research carried out by the applicant, and be limited to ½ hour with a further ½ hour discussion period.

3. The Secretary of the Scientific Section for Anaesthesia must be notified of intention of competing for this prize by 30 June 1961, and the completed paper must be in the Secretary's hands by 31 July 1961.

4. The prize will be awarded on the decision of a panel of judges appointed by the Chairman of the Anaesthetic Section, and if the number of entries warrants it, this panel will select only those papers which it considers to be of the highest standard for presentation at the Congress.

5. Three factors will be taken into account in awarding the prize: the subject matter and format of the paper, the manner of its presentation before a meeting, and the ability of the author to handle a discussion period on his paper by the meeting.

6. All entries must be accompanied by a letter from the head of the anaesthetic department concerned certifying the status of the entrant.

AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENT

SUID-AFRIKAANSE ONDERLINGE MEDIESE HULPVERENIGING

Lede van die Vereniging word nogmaals daaraan herinner dat onderhandelinge met die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging afgebreek is, en dat die goedkeuring teruggetrek is wat aan groepe verleen word wat deur die Hulpvereniging geadministreer word.

Soos tevore vermeld, is dit die beleid van die Mediese Vereniging om die ontwikkeling van mediese hulpverenigings aan te moedig. Die goeie bedoelings van die Vereniging om die voordeel van die voorkeurtarief uit te brei na persone wat kon kwalifiseer vir lidmaatskap van 'n mediese hulpvereniging, is weerspieël in die onderhandelinge wat met mediese versekeringskemas aangegaan is. Hulp is aan hulle verleen om groepe te stig wat kon voldoen aan die reëls waaraan mediese hulpverenigings moet voldoen om goedkeuring te ontvang.

Die voorkeurtarief van die Vereniging was bedoel alleen vir lede van goedgekeurde mediese hulpverenigings, m.a.w. vir persone wat aan 'n bepaalde inkomstegroep behoort. Die Vereniging kon egter nie instem dat persone met inkomste bo daardie groep in staat moes wees om behandeling teen die voorkeurtarief te eis nie.

Nietemin, terwyl bogenoemde onderhandelinge nog aan die gang was, het die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging begin om tjeks aan dokters aan te bied vir bedrae wat aansienlik minder was as die bedrae van die rekenings wat aan hul pasiënte gelewer is. Hierdie tjeks is gekodifieerde tjeks genoem en is *in volle vereffening* van die rekenings aangebied. Dit kom daarop neer dat die Hulpvereniging 'n poging aangewend het om geneeshere te oorreed om gelde op die basis van die voorkeurtarief aan te neem ter betaling van rekenings wat volgens die gelde vir private pasiënte gelewer is. Dit was teenstrydig met die besluit van Federale Raad (waarvan die Hulpvereniging ten volle bewus was) wat verlang dat versekeringskemas enige voordele direk aan daardie pasiënte wat nie vir die voorkeurtarief kwalifiseer nie, sal betaal. Alhoewel die Hulpvereniging gewaarsku is dat die Vereniging nie kon toestem tot die uitgee van hierdie gekodifieerde tjeks nie, en dat die voortgesette gebruik daarvan tot beëindiging van onderhandelinge met die Hulpvereniging kon lei, het die Hulpvereniging aangedui dat dit nietemin sou voortgaan met die uitgee van hierdie gekodifieerde tjeks ter betaling van rekenings vir mediese dienste wat aan sy lede gelewer word.

Die Vereniging was geen ander keuse gelaat nie as om alle onderhandelinge met die Hulpvereniging te beëindig. Lede van die Vereniging behoort dus alle tjeks wat hulle ontvang, uitgemaak vir 'n bedrag minder as dié van die rekening aan die pasiënt gelewer, aan die Hulpvereniging terug te stuur.

Plaza-gebou 28
Banklaan
Pretoria
8 Februarie 1961

L. M. Marchand
Medesekretaris

SOUTH AFRICAN MUTUAL MEDICAL AID SOCIETY

Members of the Association are once more reminded that negotiations with the South African Mutual Medical Aid Society have been broken off and that the approval which had been granted to certain groups administered by the Society has been withdrawn.

As has been mentioned before, the policy of the Medical Association is to encourage the development of medical aid societies. The good intentions of the Association to extend the benefit of the preferential tariff to persons who would qualify for membership of a medical aid society were carried out by the negotiations which were conducted with medical insurance schemes. Assistance was given them to establish groups which could comply with the rules to which medical aid societies had to conform in order to receive recognition.

The preferential tariff of the Association was intended to apply only to members of approved medical aid societies, in other words, to persons who belonged to a particular income category, but the Association could not agree that persons with an income above that category should be able to claim treatment at the preferential tariff.

However, while the abovementioned negotiations were still in progress, the South African Mutual Medical Aid Society began tendering cheques to doctors for amounts substantially lower than those of the accounts rendered to their patients. These cheques were termed 'coded cheques' and were tendered *in full settlement* of the accounts. This meant in effect that the Society was endeavouring to persuade medical practitioners to accept fees on the basis of the preferential tariff in payment of accounts rendered at private rates. This was in conflict with a resolution adopted by Federal Council, of which the Society was fully aware, requiring insurance schemes to pay any benefits direct to those patients who did not qualify for the preferential tariff. Although the Society was warned that the Association could not agree to these 'coded cheques' being issued and that their continued use would lead to the breaking off of negotiations with the Society, the Society indicated that it would nevertheless continue to issue these 'coded cheques' in respect of its members in payment of medical services rendered to them.

The Association was left with no alternative but to terminate all further negotiations with the Society. Members of the Association should therefore return to the Society all cheques received by them which are made out for an amount less than that of the account rendered to the patient.

28 Plaza Building
Bank Lane
Pretoria
8 February 1961

L. M. Marchand
Associate Secretary

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

Following is a list of international medical meetings to be held during the period July - December 1961. Alterations and additions to this list will continue to be published in the *Journal* from time to time together with information concerning meetings taking place before July 1961. For the full list of meetings taking place between January - June 1961 see the issue of the *Journal* for 30 July 1960 (**34**, 658).

First International Congress of Rural Medicine (Congrès International de Médecine Agricole), Tours, France, 2 - 9 July 1961. Prof. Vacher, Secrétaire Général, c/o Institut National de Médecine Agricole, Tours (Indre-et-Loire), France.

European Organization for Research on Fluorine and Dental Caries Prevention, 8th meeting, London, 5 - 8 July 1961. Dr. J. R. Forrest, Senior Dental Officer, Ministry of Health, Savile Road, London, W.1.

International Ophthalmic Optical Congress, London, 5 - 12 July 1961. G. H. Giles, President, International Optical League, 65 Brook Street, London, W.1.

Conference on Optical Instruments and Techniques, London, 10 - 14 July 1961. K. J. Habell, National Physical Laboratory, Teddington, Middlesex, England. International Commission for Optics and British National Committee for Physics.

Third International Congress of Dietetics, London, 10 - 14 July 1961. Miss D. F. Hollingsworth, British Dietetic Association, 251 Brampton Road, London, S.W.3. International Interim Committee of Dieticians.

International Diabetes Federation, 4th Congress, Geneva, 10 - 14 July 1961. Dr. B. Rillet, Secretary General of Federation, 4 boul. des Tranchées, Geneva, Switzerland.

Seventh International Congress of Life Insurance Medicine, Lisbon, 15 - 18 July 1961. Dr. Lopo de Carvalho Cancella, Secretary, Parede, Portugal. Dr. F. Kaufmann, General

Secretary, Permanent International Committee for Life Insurance Medicine, Genferstrasse 35, Geneva 2, Switzerland.

Ciba Foundation Symposium on Pulmonary Structure and Function, London, 19 - 21 July 1961. Ciba Foundation, 41 Portland Place, London, W.1 (by invitation).

Latin Federation of Medical Electro-radiological Societies, 5th Congress, Paris, 22 - 29 July 1961. Dr. Charles Proux, Secretary, Fédération Latin des Sociétiés d'Électro-radiologie Médicale, 9 rue Daru, Paris 8e, France.

Seventh International Congress of Otolaryngology, Paris, 23 - 28 July 1961. Dr. H. Guillon, Secretary General, 6 av.Mac-Mahon, Paris 17e, France.

Twelfth International Congress of Urology, Rio de Janeiro, 24 - 30 July 1961. Dr. J. Silva de Assis, Secretary, P.O. Box 1275, Belo-Horizonte, Brazil. Prof. René Küss, Secretary, International Society of Urology, 152 Av. Victor Hugo, Paris 16e, France.

Ciba Foundation Symposium on Immunological Developments in Research on Protein and Polypeptide Hormones, London, 26 - 28 July 1961. Ciba Foundation, 41 Portland Place, London, W.1 (by invitation).

International Society of Obstetrical Psychoprophylaxis, 1st Congress, Paris, July 1961. 31 rue St. Guillaume, Paris 7e, France.

International Union of Biological Sciences, 14th General Assembly, Amsterdam, July 1961. Prof. G. K. Stebbins, Secretary-General, c/o Department of Genetics, University of California, Davis, California.

International Congress on Pharmacology, Stockholm, 3 - 10 August 1961. Prof. Börje Uvnäs, Pharmacology Institute, Karolinska Mediko-kirurgiska Institutet, Solnavägen 1, Stockholm, Sweden.

Fifth International Congress of Biochemistry, Moscow, 10 - 16 August 1961. Dr. N. M. Sissakian, Secretary-General of Congress, Leninsky prospekt 33, Moscow B-71, USSR.

Second International Medical Conference on Mental Retardation, Vienna, 14 - 19 August 1961. Dr. O. Stur, Univers. Kinderklinik, Lazarettgasse 14, Vienna 9, Austria.

Fifth International Congress of Psychotherapy, Vienna, 21 - 26 August 1961. Dr. W. Spiel, Lazarettgasse 14, Vienna 9, Austria. International Federation for Medical Psychotherapy.

First International Pharmacological Meeting, Stockholm, 22 - 25 August 1961. Dr. Arvid Wretlind, Secretary General, Karolinska Institutet, Stockholm 60, Sweden. International Union of Physiological Sciences, Section on Pharmacology.

European Society of Haematology, 8th Congress, Vienna, 28 August - 2 September 1961. Dr. H. Fleischhracker, Secretary General of Congress, Frankgasse 8, Billrothhaus, Vienna IX, Austria.

Sixth International Congress on Mental Health, Paris, 30 August - 6 September 1961. World Federation for Mental Health, 19 Manchester Street, London, W.1.

International Congress of Exfoliative Cytology, Vienna, 31 August - 2 September 1961. Office of the Secretary of Congress, 666 Elm Street, Buffalo 3, N.Y. International Academy of Gynecological Cytology. Precedes 3rd World Congress of Gynecology and Obstetrics, 3 - 9 September 1961.

Meeting on Preventive and Social Medicine, Evian, France, 31 August - 4 September 1961. Société Française de Médecine Préventive et Sociale, 1 rue de Courcelles, Paris 8e, France.

International College of Angiology, Congress, August 1961. Prof. Paessler, Vegelsfelden Ass 26, Leverkusen, Germany.

International Union of Professional Gynaecologists and Obstetricians, 5th Congress, Vienna, late August 1961. Dr. J. Courtois, Secrétaire Général Permanent, Union Professionnelle Internationale des Gynécologues et Obstétriciens, 1 rue Racine, Saint-Germain-en-Laye (S.-et-O.), France. Precedes 3rd World Congress of Gynecology and Obstetrics, 3 - 9 September 1961.

International Society of Surgery, 19th Congress, Dublin, 2 - 9 September 1961. Dr. T. C. J. O'Connell, 35 Fitzwilliam Place, Dublin, Ireland. Precedes 5th Congress of International Cardiovascular Society, 7 - 9 September 1961.

Third World Congress of Gynecology and Obstetrics, Vienna, 3 - 9 September 1961. Prof. Tassilo Antoine, President, c/o Universität-Frauenklinik, I, Spitalgasse 23, Vienna 9, Austria.

Seventh Inter-American Congress of Radiology, São Paulo, 3 - 10 September 1961. Dr. Walter Bomfim-Pontes, Secretary General, Rua Cesario Motta 112, São Paulo, Brazil.

Fourth International Congress of Neuropathology, Munich, 4 - 7 September 1961. Prof. Hans Jacob, Secretary, Marburg an der Lahn, Germany.

Tenth International Congress on Rheumatic Diseases, Rome, 4 - 7 September 1961. Prof. C. B. Ballabio, c/o Clinica Medica, Via F. Sforza 35, Milan, Italy.

Symposium on Elimination of Infectious Diseases Affecting Laboratory Animal Colonies, Prague, 4 - 8 September 1961. International Committee on Laboratory Animals, Dr. W. Lane-Petter, Secretary General, c/o M.R.C. Laboratories, Woodmansterne Road, Carshalton, Surrey, England. In conjunction with session of the International Committee on Laboratory Animals.

Fourth International Congress of Angiology, Prague, 4 - 9 September 1961. Dr. Gerson, Secretary General, 4 rue Pasquier, Paris 8e, France.

Ninth International Congress of the International Union of Railway Medical Services, Istanbul, 5 - 9 September 1961. Dr. J. Ortega, 13 rue de Château, Landon, Paris 10e, France.

International Cardiovascular Society, 5th Congress, Dublin, 7 - 9 September 1961. Dr. H. Haimovici, Secretary General of the Society, 715 Park Avenue, New York 21, N.Y.

(to be continued)

DIE LIEFDADIGHEIDSFONDS : THE BENEVOLENT FUND

Met dank word die volgende skenkings gedurende die maand Januarie 1961 erken:

The following donations during January 1961 are gratefully acknowledged:

Geloftekaarte ter Nagedagtenis aan: Votive Cards in Memory of:

Dr. E. J. Swirsky *by* Dr. I. J. Zadikoff; Mrs. S. J. Oliphant *by* Shirley Cole; Dr. H. Pearson *by* Drs. Botha, E. Hafner, J. A. Richter and Ferguson; Mr. A. Hesse *by* Dr. and Mrs. R. Theron; Oorlede Moeder van mnr. en mevr. H. Dumas *deur* Dr. J. J. van Zyl; and Bernand and Anne Doctor *by* Dr. H. L. Goldblatt.

Totaal Ontvang van Geloftekaarte: £8 18s. 6d.
Total Received from Votive Cards:

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Mevr. Nieuwoudt *deur* Dr. H. Muller, Dr. P. W. Bosman, Dr. H. W. Clegg, Dr. A. S. Peden and H. Rifkin.

Dr. G. Rosendorff *by* Mr. Arnold Katz and Dr. Jack Levin.

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Totaal Ontvang van Dienste Gelewer: £48 7s. 0d.
Total Received from Services Rendered:

Donasies: Donations:

	£	s.	d.
Dr. L. C. Goument	10	10	0
Dr. W. M. S. Basson and Dr. Lubbe	10	0	0
Drs. Harries, Kuschke and Hofmeyr	1	1	0
Southern Transvaal Branch (M.A.S.A.)	2,454	17	9

Totaal Ontvang van Donasies: £2,476 8s. 9d.
Total Received from Donations:

Groot Totaal: Grand Total £2,533 14s. 3d.

IN DIE VERBYGAAN : PASSING EVENTS

Important. Will members please note that contrary to the information published in the *Journal* of 11 February (**35**, 116), the venue for the Extraordinary General Meeting of the Association will be the **Festival Hall, Maitland Hotel, Maitland Street, Bloemfontein,** and *not* Johannesburg. This meeting has been called to consider the implementation of the Medical Services Plan in all Provinces of South Africa, and will be held on 8 March 1961 at 10 a.m.

Belangrik. Die aandag van lede word gevestig op 'n verandering van die inligting gepubliseer in die *Tydskrif* van 11 Februarie (**35**, 116). Die vergaderplek van die Buitengewone Algemene Vergadering van die Vereniging is verander na die **Festival Hall, Maitland-hotel, Maitlandstraat, Bloemfontein,** en sal *nie* in Johannesburg gehou word nie. Hierdie vergadering is saamgeroep om die instelling van die Mediese Diensplan in al die Provinsies van Suid-Afrika te oorweeg, en sal om 10 vm. op 8 Maart 1961 gehou word.

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday, 27 February at 5.10 p.m. in the Institute Lecture Theatre. Dr. A. R. P. Walker will speak on 'Some aspects of research work bearing on coronary heart disease undertaken on local White and non-White populations'.

* * *

Dr. R. Geerling has joined Dr. Frances Reinhold in neurological and psychiatric practice at 617 Pan Africa House, 77 Troye Street, Johannesburg, (telephone 22-8631, not in current Telephone Directory). Dr. Geerling has recently retired from the staff of the Tara Hospital, Johannesburg, and, after a vacation trip to Europe, has now entered private practice.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 22 February at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Prof. L. N. Pyrah, Professor of Urological Surgery, Leeds University, will speak on 'Surgery of the colon in relation to the urinary tract'. All members of the Medical Association are welcome to attend this lecture.

* * *

Dr. J. Abelsohn, anaesthetist, of Cape Town, has resumed his practice after a recent overseas visit.

* * *

Southern Transvaal Branch (M.A.S.A.). The Annual General Meeting of this Branch will be held on Tuesday 21 February at 8.15 p.m. at Medical House, 5 Esselen Street, Hospital Hill, Johannesburg. In addition to the usual business of the Annual General Meeting, the Branch will debate a motion proposed by the Branch Council for adoption, namely 'That the Southern Transvaal Branch of the Medical Association of South Africa agrees to sponsor an appeal for funds for the purpose of establishing a Clinical Research Unit in the proposed new Medical School building of the University of the Witwatersrand'.

* * *

Dr. Bernard Levinson, M.B., B.Ch., D.P.M. (Rand), will commence practice as a psychiatrist on 1 March 1961 at 504 Osler Chambers, Jeppe Street, Johannesburg. Telephones: Rooms 23-9860, residence 42-9686.

Department of Anaesthesia, University of Cape Town. On Saturday 25 February a meeting will be held in the A-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, at 9.30 a.m. Dr. W. Lambrechts will speak on 'Thank you, Lord Nuffield'. Interested doctors are invited to attend this meeting.

* * *

Prof. L. N. Pyrah, Professor of Urological Surgery, Leeds University, and Hon. Director of the Medical Research Council Unit for research in metabolic disturbances in surgery, will deliver a lecture on 'The renal calculus — some aspects of aetiology and treatment (including hyperparathyroidism)' on Tuesday 21 February at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Although this lecture is primarily intended for physicians, all medical practitioners who are interested are invited to attend the lecture.

OBITUARY — DR. HEINZ LORD

Dr. Heinz Lord the newly appointed Secretary General of the World Medical Association, who replaced Dr. Louis H. Bauer who retired from the position at the end of last year, died suddenly after a heart attack on 3 February. He was 43 years of age.

Dr. Heinz Lord

Dr. Lord had an interesting international background. Born a Peruvian citizen, he was brought up and educated in Hamburg, Germany, and Zurich, Switzerland. During the Second World War, being anti-Nazi, he was placed in a concentration camp. Near the end of the war he was on board a steamship, containing 800 internees, which was bombed in the Baltic Sea. Dr Lord was one of 28 survivors. Returning to Hamburg at the end of the war, he was active in organizing the Marburger Bund.

In 1954 he migrated to the United States. Following 3 years of further postgraduate training, he entered surgical practice in Barnesville, Ohio. He was a member of the American Medical Association, a Fellow of the International College of Surgeons and a member of the German Urological Society.

BOEKBESPREKINGS : BOOK REVIEWS

ORTHOPAEDIC SURGERY

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery — Upper Extremity. By Rodolfo Cosentino, M.D. Pp. xiv + 192. 134 figures. 84s. Oxford: Blackwell Scientific Publications Ltd. 1960.

Stimulated by no less a personality than Arthur Steindler, Dr. Cosentino's treatise on anatomy and approaches of orthopaedic operative procedures will serve a real need in the field of orthopaedic surgery. It has become customary in describing approaches in operative orthopaedic surgery to limit description to the wound and the approach to the particular piece of bone or joint.

Dr. Cosentino has approached the problem from the wider anatomic concept and serves to remind us how vastly important the gross anatomy is in the environment of our surgical procedure. His illustrations are beautifully clear and anatomically accurate, thus offering the surgeon a quick

reference book which could well afford to be kept on the shelves in close proximity to the surgical theatre itself. As such, the book will be made use of not only by younger, relatively inexperienced men, but will also prove valuable to the surgeon of standing. Quick anatomic revision before operating cannot be too often emphasized.

Of recent times the orthopaedic surgeon has adapted himself to numerous approaches, and this fact requires him to make constant revision of gross anatomy. Likewise, with the age of increasing trauma this book will find a very valuable place in the field of orthopaedic surgery. C.E.L.A.

CLINICAL ENDOCRINOLOGY

Clinical Endocrinology. I. Edited by Edwin B. Astwood, M.D. Pp. xvi+724. Figures. $18·75. New York and London: Grune & Stratton, Inc. 1960.

This is a book of essays on selected topics in endocrinology by a large number of different authors. It is essentially on the same lines as the British 'Modern Trends' series. Such volumes are extremely difficult to review, since naturally the standard is variable and the very different topics appeal to different people. For example, an interesting contrast is offered in the section on reproduction, where Turner describes the syndrome which bears his name in a chapter whose latest reference is dated 1947, while Grambach hardly cites a work earlier than 1958. Incidentally the essay by Grambach is extremely comprehensive and clear.

There is just a scattering of non-American authors, mainly from Scotland and Australia. Among these we find Nordin, with an interesting, but highly speculative, discussion on metabolic bone disease — rather too brief to be of great value. Dunlop considers the oral hypoglycaemic agents, but one feels that his experience of these has been rather limited. Hall, from Australia, has the chapter on gynaecomastia which seems rather uninspired and omits consideration of perhaps the most important facet of the subject — the prevalence of this condition in large areas of Africa. An author from this continent would have seemed a better choice.

The book is nicely produced and should be obtained by all libraries and by anyone else who likes reading short essays by authorities in their own fields. W.P.U.J.

A BOOK FOR MOTHERS

Babies and Young Children. Feeding: management: care. 2nd edition. By Prof. Ronald Illingworth and Dr. Cynthia Illingworth. Pp. vii + 331. 24 photographs and 50 pen sketches. English price 18s. net. London: J. & A. Churchill Ltd. 1960.

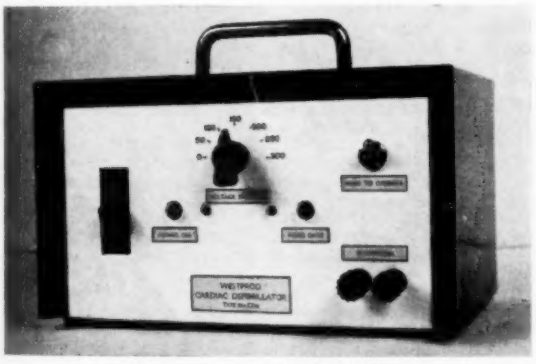
The first edition of this book proved sufficiently popular and useful to make a reprinting necessary, and this second edition has resulted in a thorough revision of the text with additions, subtractions and rearrangement of chapters.

The information given is based on the many questions put by mothers at consultations, all of which have been carefully noted at the time, so that the book is designed to give complete coverage of all aspects of child care, particularly on those matters which so often worry mothers.

The pen sketches are both instructive and entertaining, and there are 24 photographs, which will have a wide appeal. It is written for mothers and can be recommended to them. A.H.T.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

Westprod Cardiac Defibrillator



Westprod Cardiac Defibrillator

Westdene Products (Pty.) Ltd. announce the introduction of the Westprod Cardiac Defibrillator, manufactured by E. J. Middleton Electronics (Pty.) Ltd. of Johannesburg, and supply the following information:

The Westprod Cardiac Defibrillator has an operating voltage of 220/250 volts (A.C.); the output on the patient circuit is 50, 100, 150, 200, 250 and 300 volts and the impulse to the patient is of 0·2 seconds duration. A non-locking switch allows impulses to be repeated as required. There is a safety automatic relay switch in the patient's circuit for complete protection. There are two electrodes, one is spoon-shaped and the other is flat and round. Both are on insulated handles (not illustrated). 'Power on' and 'impulse on' are indicated by neon lamps.

The Westprod Cardiac Defibrillator measures 9×6×5½ in. in size and weighs approximately 5 lb. It is fitted with a carrying handle and is mounted on rubber feet. The Defibrillator is housed in red metal cabinet for easy identification. (South African Bureau of Standards report available for inspection on request.)

Further information may be obtained from Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

BRIEWERUBRIEK : CORRESPONDENCE

INCOME CEILING OF £2,300

To the Editor: In support of Mr. M. H. Luntz's letter[1] in the *Journal* of 14 January 1961 I should like to have this letter published in your Correspondence Columns.

We are repeatedly told that Federal Council has as its main function the safeguarding of the interests of the profession, yet we are now told officially that the Executive Committee has agreed to accept an income ceiling of £2,300 *per annum* for members of insurance company-sponsored medical aid societies. I feel that those members of Federal Council who have approved this on behalf of the medical practitioners of this country are not in touch with reality. What, for instance, in a poor town, is the percentage of population earning in excess of this figure?

There are many doctors, I am sure, who would be happy to earn a clear £2,300 *per annum*, with an adequate pension and a paid holiday, and paid sick-leave thrown in, yet they are now being asked to give these wealthy executives, business and commercial men, subsidized services. Has Federal Council, for example, arranged any reciprocity for us? Will I be able to have a bank overdraft at a reduced rate of interest? I feel that many true medical aid schemes will soon switch to insurance schemes, in order that more members may be covered, and it will not then be long before bank managers, etc. will be entitled to 'cut rates'.

One wonders what the percentage of patients in the average practice is, whose income is in excess of £2,300 *per annum*. We are now developing a situation as envisaged by Federal Council, of reduced rates for the upper income class, while the poor junior apprentice or artisan who is not yet eligible for medical aid society membership, is being charged full fees. Is this just or fair?

The average general practitioner who does not undertake

surgery, would have to see at least 24 patients a day for 25 days a month at this 'cut rate' to earn a clear £2,300 *per annum*, and that, without a holiday or pension. According to the Council, then, he would still be classed as a poor man requiring assistance — if he were non-medical. Being a doctor his own peers feel he can economically afford to give a concession in the matter of fees.

To conclude, I would suggest that the time has come for members of the Association to get together and refuse to have any dealings with any insurance company-sponsored medical aid scheme, and refuse solidly to be dictated to by any 'big business' group. Let us retain the right to set a fair fee for our services.

Strelson House
44 Union Street
East London
30 January 1961

Frank K. Gutsche

1. Correspondence (1961): S. Afr. Med. J., **35**, 40.

BURIED SILK SUTURES IN LENS SURGERY

To the Editor: I read with interest Mr. M. H. Luntz's article 'An assessment of buried silk sutures in lens surgery'.[1]

While I agree with Mr. Luntz about the value of buried silk sutures, I do not feel that he proves his case by the figures quoted. The one series was operated upon by a single surgeon, while the other series is made up of the composite figures of 12 surgeons. The nursing care, too, was different, and in view of the great importance of nursing in cataract cases it is important to have a uniform standard of nursing care in any comparative series.

I wish, however, to lend my support to Mr. Luntz's view that buried silk sutures are of great help in cataract surgery. The fact that they need not be removed is greatly appreciated by the patients.

S. Etzine

815 Philadelphia Corner
Jeppe Street
Johannesburg
31 January 1961

1. Luntz, M. H. (1961): S. Afr. Med. J., **35**, 61.

CARBOCAINE POISONING

To the Editor: We think Dr. L. G. R. van Dongen and Dr. H. Glietenberg should be congratulated on their candour in publishing 'A case of toxicity to excess "carbocaine".'[1] In our opinion, this case can be described more accurately, although less euphemistically, simply as 'intoxication'. Goodman and Gilman[2] state, '. . . it is highly significant that all the local anaesthetics produce the same type of symptoms when given in toxic amounts'. Daniel Moore, author of 2 excellent books on regional blocks[3,4] noted that procaine and lignocaine overdosage may lead to central depression without the minor signs of cerebral stimulation progressing to convulsions and peripheral vascular collapse . . . that being the common pattern for all others, i.e., cocaine, 'nupercaine', cyclaine, amethocaine, 'metycaine' and certainly also carbocaine.

From January 1948 to December 1959, Moore and his colleagues performed 36,113 regional block procedures.[5] They treated 430 minor systemic toxic reactions with oxygen alone. This treatment, not unknown to anaesthetists, invariably succeeds in supplying the manifold increase in the oxygen requirement of the cortex and medulla which would otherwise lead to convulsions and which accompany the unmistakable early signs and symptoms of systemic toxic reaction. These are: restlessness, nervousness, apprehension, pugnaciousness, unreasonableness, loquacity, incoherent speech, headache, dizziness, blurred vision, metallic taste, roaring in the ears, nausea and vomiting, choreiform movements, tremors and twitching.[5] *Only if these warnings are disregarded by allowing cerebral anoxia to develop will convulsions ensue.*

Of Moore's patients mentioned above, 10 manifested unconsciousness and apnoea as the only signs of a severe systemic toxic reaction. Here again oxygen only was required, this time by intermittent-positive-pressure artificial respiration. These patients recovered in 10 - 20 minutes after which time they submitted to the intended surgery without sequelae. This series included 93 severe toxic reactions with convulsions. The convulsions of 84 were stopped by the administration of oxygen. Of the remaining 9 patients, 5 had their convulsions before 1956, and received intermittent 50-mg. doses of intravenous thiopentone; they remained unconscious for 2 - 3 hours. The remaining 4 patients were treated after 1956 and received 40 mg. suxamethonium intravenously; they responded after 10 - 15 minutes and were fully recovered 30 - 45 minutes after the convulsions began. One of us mentioned suxamethonium as the remedy for convulsions before the publication of Moore's series just discussed.[6] Thus, to quote Moore:[5] reactions were a result not of allergy, but of high concentrations of drug in the blood; barbiturates used to treat such reactions may do harm rather than good and may actually hasten the death of a patient; barbiturates given pre-operatively did not prevent reaction; correction of oxygen want was the key to successful treatment; and *suxamethonium, not a barbiturate, is now the drug of choice to stop convulsions and adequately oxygenate the patient.* We can mention in passing that the same principle has been used successfully in the treatment of status epilepticus with 'tubarine' and artificial respiration. Like thiopentone, 'methedrine' and allied stimulants should also be condemned, certainly at least until full oxygenation has been accomplished. It is clear why one of us recently witnessed ventricular fibrillation (in another city) immediately after the intravenous administration of methedrine in an anoxic, hypotensive patient subsequent to a lumbar epidural block.

Finally, we should like to comment on the intriguing implication that the carbocaine overdosage led to reactivation of rheumatic fever. When an animal is injured, develops an infection, is exposed to X-rays or to cold, or is subjected to almost any circumstances not ordinarily encountered in its day-to-day existence, a series of bodily reactions take place. Selye suggested the balance can thus be disturbed between the amounts secreted of mineralocorticoid inflammation-stimulating hormones, such as DOC (deoxycortone), and of glucocorticoids such as cortisone. The DOC did actually, once, at least, aggravate the arthralgia in a patient with pre-existing rheumatoid arthritis who subsequently developed Addison's disease.[7]

Similarly, we are not surprised that Dr. van Dongen's patient developed signs of stress; it is remarkable that she survived, and the authors must be complimented on their prompt and successful treatment, and their keen subsequent interest in the reaction. After all, anaesthetists reported in the *Journal* during 1957 that they had injected 7 ml. of 2% lignocaine (140 mg.) without adrenaline in a 24-month old, 28-pound child! The subsequent reaction was considered worthy of reporting, although it was so obviously a gross overdose! That is all the more lamentable, when it is remembered that non-specialists administer the vast bulk of local anaesthetics in this country. Why it is that overdosage of local anaesthetics is not taken as seriously as overdosage with arsenic is a complete mystery to us. Surely such abuse is the reason why patients are so frequently deprived of a clearly safer anaesthetic method such as caudal, lumbar epidural, or subarachnoid spinal block, particularly in obstetrics, as Dr. van Dongen and Dr. Glietenberg themselves point out.

J. W. Mostert,
L. Milner,
J. L. du Preez,
A. P. de Villiers

Department of Anaesthetics
University of the Witwatersrand
Johannesburg
4 February 1961

1. van Dongen, L. G. R. and Glietenberg, H. (1961): S. Afr. Med. J., **35**, 73.
2. Goodman, L. and Gilman, A. (1955): *Pharmacological Basis of Therapeutics*. New York: Macmillan.
3. Moore, D. C. (1957): *Regional Block. A Handbook for Use in the Clinical Practice of Medicine and Surgery*. Springfield: Thomas.
4. *Idem* (1955): *Complications of Regional Anaesthesia: Etiology — Signs — Symptoms — Treatment*. Springfield: Thomas.
5. Moore, D. C. and Bridenbaugh, L. D. (1960): J. Amer. Med. Assoc., **174**, 842.
6. Mostert, J. W. (1960): Brit. J. Anaesth., **32**, 334.
7. Leading Article (1954): Brit. Med. J., **1**, 1195.